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MALIGNANT HYPERTENSION*

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INTRODUCTION

MUCH has been written in the recent years concerning a rapidly fatal form of hypertension termed by various authors "malignant hypertension"[1], "malignant nephrosclerosis"[2], "arterionecrosis"[3], "arteriolonecrosis"[4], and "malignant phase of essential hypertension"[5]. A survey of this literature reveals important differences in the descriptions of the clinical picture and more particularly of the underlying pathology in this type of hypertension. It was therefore considered desirable to study the postmortem material obtained from patients in whom the diagnosis of malignant hypertension had been made during life. This paper represents the result of such a study.

Clinically, malignant hypertension manifests itself by a history of severe headache, weight loss, weakness, and visual disturbances. Examination of patients with these symptoms reveals severe persistent hypertension and a typical retinal picture. The retina in malignant hypertension has been described by Wagener[3], and others[9, 10, 11, 12]. It has been termed "renal retinitis"[9], "retinitis of malignant hypertension"[8], "albuminuric retinitis"[10], "malignant hypertensive neuroretinitis"[11], and "hypertensive neuroretinopathy"[5]. Edema and hyperemia of the optic discs are the outstanding features. In addition, sclerosis of the arterioles, retinal hemorrhages, and "cotton wool" spots are present.

The earlier[1, 2, 3, 4, 5] authors stressed the invariable terminal occurrence of acute uremia in patients with malignant hypertension. On the other hand, Keith, Wagener, and Kernohan[6] and Murphy and Grill[7] emphasized the failure of other organs particularly the heart and brain, in such patients, and described cases in which there was absolutely no evidence of renal insufficiency. Volhard and Fahr[1], Stern[3], Herxheimer[4], and Fishberg[5] regard the fundamental pathology of malignant hypertension to consist in renal necrotizing arteriolitis, while Keith, Wagener, and Kernohan[6], described only a very widely distributed arteriolar medial hypertrophy. Murphy and Grill[7] observed little medial hypertrophy; but they did find renal arteriolitis in some instances but could demonstrate no correlation between its presence and the occurrence of renal insufficiency.

There has, however, always been universal agreement that the elevation of blood pressure in malignant hypertension is of the *primary essential type.*

Analysis of the cases seen at the Beth Israel Hospital, Boston, with the clinical picture of malignant hypertension as described by Keith, Wagener, and Kernohan[6] revealed the fact that the hypertension in some of them was unquestionably of secondary origin. This was discovered only at postmortem examination and was entirely unsuspected during life. Moreover, even in those instances in which the hypertension was undoubtedly primary, autopsy revealed a variety of underlying pathological processes. Five cases typical of the series under the direct observation of the authors, are reported.

REPORT OF CASES

CASE 1. History: I. B. (B. I. H. No. 4749), a forty-three year old Russian Jewish iron worker with a family history of heart trouble in one sister and with a past history of measles in childhood, occasional slight epistaxis from the left nostril since childhood, several sore throats each winter, and nocturia of four or five times a night for about four years, entered the hospital on December 13, 1929, complaining of weakness of two weeks' duration. About four weeks before admission the patient noted loss of appetite, dyspnea while at work, spells of dizziness, and a slight cough, the last most marked at night. These symptoms progressed and the patient took to bed two weeks before admission. At that time he also became orthopneic and his cough began to be associated with pain in his right chest. Twelve days before admission the patient was told that he had high blood pressure. He lost twelve pounds in weight in the four months preceding admission.

Physical Examination: The patient was a dyspneic, orthopneic man with slight cyanosis of the ears and lips and Cheyne-Stokes respiration. The anterior-posterior diameter of the chest was increased. The percussion note was somewhat hyperresonant. A few moist râles were heard at the lung bases. The heart was moderately enlarged and presented an accentuated aortic second sound. The liver was palpable three centimeters below the costal border. The peripheral arteries showed marked thickening and tortuosity. The blood pressure was 225 systolic and 140 diastolic. Ophthalmoscopic examination revealed hyperemia of both discs with slight papilledema. The vessels were narrowed and tortuous and showed marked arteriovenous nicking. Both fundi contained numerous

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irregular areas of degeneration temporally and scattered punctate, linear and irregular fresh and old hemorrhages as well as areas of exudate, the last especially prominent about the maculae.

Laboratory Data: Seven urine examinations showed a specific gravity ranging from 1.002 to 1.012, with a trace to a large trace of albumin in all specimens; the sediment was negative except for the finding of a few leucocytes on one occasion and several granular casts on another. The white blood cell count ranged between 10,900 to 14,200 per cu. mm. with an increase in the polymorphonuclear cells to 82 per cent. The red blood cell count was 4,200,000 per cu. mm. with a hemoglobin of 70 per cent (Tallqvist). Blood Wassermann reaction was negative. Phenolsulphonphthalein excretion after intramuscular injection of the dye was 5 per cent after two hours and ten minutes on two occasions. The blood sugar was 95 mg. per cent. The blood nonprotein nitrogen on admission was 54 mg. and the blood creatinine was 3.6 mg. These values rose to 141 mg. and 7.1 mg. per cent respectively during the following two weeks. The icteric index was 11.3. The serum phosphate was 7.1 and 8.9 mg. per cent on two occasions toward the end of his course, and the serum calcium was 10.2 and 9.3 mg. per cent. The blood CO_2 combining power ranged between 30 and 38 volumes per cent. The blood cholesterol on two occasions was 208 and 195 mg. per cent respectively.

Electrocardiogram showed normal rhythm, rate 90, with inverted T″ and T‴ and slurred QRS′.

Clinical Course: The patient was given a salt poor 30 gram protein diet, fluids to 1500 cc., and was digitalized. His blood pressure fluctuated about 210 systolic and 120 diastolic during the first two weeks of his stay and then slowly fell to 170 systolic and 95 diastolic as the patient lapsed into coma. During the three weeks of his hospital stay he had several attacks of angina pectoris and severe epistaxes. His weakness and cyanosis increased and the patient failed rapidly, expiring on the twenty-first day of his stay.

Autopsy: (No. A30-1) Autopsy was performed by Drs. Stern and Schlesinger two hours postmortem. Both legs showed considerable edema. The eyelids were puffy. The peritoneal cavity contained 250 cc. of serous fluid. The left pleural cavity contained 750 cc. and the right 700 cc. of fluid. The surfaces of both lungs bore a few small fibrous adhesions. The pericardial cavity contained 100 cc. of clear, straw-colored fluid.

The right kidney weighed 190 and the left 200 grams. The capsules stripped with difficulty, leaving a slightly granular, pitted, pale, brownish-yellow surface. The kidneys were firm in consistency. The cortex was well demarcated from the medulla and measured 5 mm. in thickness. The kidneys appeared congested. Microscopic examination revealed a moderate increase in the interstitial connective tissue. The connective tissue was edematous and contained a small number of chronic inflammatory cells, the latter occurring in foci in some areas. The glomeruli presented varying degrees of fibrosis. A moderate number showed marked fibrosis of the tufts and some were represented only by a mass of fibrous tissue. Most of them were normal except for occasional tiny foci of polymorphonuclear cell infiltration. The glomerular capsules showed slight thickening in a few areas, but no definite crescent formation. In a few instances adhesions had formed between capsule and tuft. The larger arteries showed slight to moderate intimal thickening. Many of the arterioles were entirely normal. A very few showed medial hyalinization and thickening. In rare instances, there were small foci of arteriolar necrosis at the point at which the afferent arterioles entered their respective glomeruli. The tubules showed marked atrophy of the lining epithelium. Many of the lumina contained granular débris, masses of fibrin, and casts of various sorts. In short, the kidneys showed chronic glomerular nephritis, and rare foci of arteriolonecrosis.

The heart weighed 650 grams. The valves were all normal. The left ventricle was markedly hypertrophied measuring 3.7 cm. in thickness except at the apex where it was thinned, measuring 0.3 cm. The right ventricle measured 0.8 cm. in thickness. The circumflex branch of the left coronary artery was completely occluded and the descending branch markedly narrowed. The other arteries showed moderate atheromatous change.

The lungs were somewhat congested and presented small scattered, pneumonic areas.

The aorta was normal in size but showed diminished elasticity. There were a few scattered atheromatous plaques, one of which in the thoracic region had ulcerated leaving a denuded area measuring 10 x 6 mm.

All of the other organs showed varying degrees of congestion. The liver showed, in addition, moderate central necrosis. The arteries presented moderate sclerosis. The arterioles of the pancreas and adrenals showed slight to moderate medial thickening and hyalinization. Those of the spleen showed similar, though more marked changes, with foci of medial necrosis and thrombosis.

Comment: This middle-aged patient with a negative past history entered the hospital with a present illness characterized by loss of weight of four months' duration, dyspnea, dizziness and cough of four weeks' duration and orthopnea and pleuritic pain of twelve days' duration. Examination revealed dyspnea, orthopnea, cyanosis, slight emphysema, a few râles at the bases of the lungs, moderate cardiac enlargement, a palpable liver, marked arteriosclerosis and a blood pressure of 225 systolic and 140 diastolic. Ophthalmoscopic examination showed typical malignant neuroretinitis. There was a slight anemia. The other laboratory findings indicated marked renal impairment. His course was steadily downhill and he died in uremia three weeks after admission. Postmortem examination revealed chronic glomerular nephritis with a small amount of superimposed vascular nephritis. Rare foci of arteriolonecrosis were also observed in the kidneys. This patient with autopsy findings of chronic glomerular nephritis presented a typical picture of malignant hypertension terminating in uremia.

Case 2. History: E. R. (B. I. H. No. 14671), a twenty-two year old American Jewish housewife with a negative family history and with a past history of measles and uncomplicated scarlet fever in childhood, entered the hospital on May 26, 1932, complaining of blindness of two days' duration. The patient was accepted for life insurance one year before admission. She was entirely well until four months before admission at which time she began to experience slight fatigue, occasional frequency and enuresis, and slight blurring of vision, the last relieved by glasses. She visited a physician who told her that her blood pressure was slightly elevated. Two days before admission she suddenly began to experience marked blurring of vision,

severe frontal headache, and severe vomiting. She soon became quite blind. She also became confused and at times comatose. She lost nineteen pounds in weight in the two months preceding admission.

Physical Examination: The patient was a well-developed, poorly nourished young woman lying in bed in no distress. The heart was normal in size, with a reduplicated first sound and an accentuated second aortic sound. There was slight thickening of the walls of the peripheral arteries. The lungs, abdomen, and extremities were entirely negative. The blood pressure was 200 systolic and 140 diastolic. Ophthalmoscopic examination revealed moderate elevation of the right disc with absent cupping. The left disc outline was blurred. The retinal vessels were entirely negative. There was a single hemorrhage above the right disc.

Laboratory Data: Eight urine examinations revealed a specific gravity ranging as high as 1.034. All the specimens showed slight or very slight traces of albumin; several contained occasional leucocytes and rare erythrocytes and granular casts. The red blood cell count on admission was 4,650,000 per cu. mm. and the hemoglobin was 75 per cent (Tallqvist). These values slowly fell to 4,120,000 per cu. mm. and 70 per cent, respectively. The white blood cell count ranged between 11,000 and 20,300 per cu. mm. with a polymorphonuclear cell count of 81 to 85 per cent. The blood sugar was 105 mg., blood nonprotein nitrogen 29 to 38 mg., blood creatinine 1.6 mg. per cent, and the blood CO_2 combining power 57 to 61 volumes per cent. The blood Wassermann, Kahn and Hinton reactions were negative. Phenolsulphonphthalein excretion after two hours and ten minutes following intramuscular injection of the dye was 50 to 55 per cent. Basal metabolism was plus 11 per cent. Lumbar puncture revealed an initial pressure of 210 mm. of water with normal dynamics. Examination of the spinal fluid revealed one cell, no globulin, a negative Wassermann reaction and a total protein of 50 mg. per cent.

X-rays of the skull and kidneys were negative.

Clinical Course: The patient was given a salt poor 40 grams protein diet with fluids to 3000 cc. During her first day she received magnesium sulphate by mouth and intramuscularly every four hours, and was also subjected to a venesection of 275 cc. After several hours her vision became normal. The next day her optic discs both showed cupping and definite margins. At this time a tiny hemorrhage and several patches of exudate were also noted. Arteriovenous nicking developed during the following week. In the course of her ten-day stay her blood pressure fell to 170 systolic and 120 diastolic. She continued to receive 50 per cent solution of magnesium sulphate by mouth in smaller doses. She was discharged on her tenth day with orders to continue the above régime at home. She was quite well for one week, and then weakness, anorexia, frequent vomiting, and attacks of pain with paresthesias in the arms supervened. At this time her blood pressure was 180 systolic and 140 diastolic. During the second week following discharge she suddenly lost the sight of her right eye. The blindness slowly improved. Thereafter, for the next two months, she was fairly comfortable except for irregular fever, occasional headaches, and pains in various parts of her body. Two and one half months after discharge, she suddenly became completely blind, and had seven bouts of left-sided convulsions over a period of several hours. Examination at this time revealed the absence of all deep reflexes. After two days she reverted to her previously fairly comfortable state. Her weakness and anorexia became worse and in the four and one-half months after discharge she lost twenty-four pounds in weight. She therefore reëntered the hospital on October 23, 1932.

Second Admission Physical Examination: At this time the examination revealed the same findings as on the first admission except that there was marked wasting. The blood pressure was 215 systolic and 150 diastolic. Ophthalmoscopic examination revealed blurring of the disc margins and absence of cupping. The arterioles were narrowed and slight arteriovenous nicking was present. There were many patches of exudate and one hemorrhage.

Laboratory Data: Fourteen urine examinations showed a maximum specific gravity of 1.016. More albumin and a greater number of leukocytes were present in all specimens than during the first admission. The blood nonprotein nitrogen was 31 mg. and the blood creatinine was 1.3 mg. per cent. The red blood cell count on four occasions varied between 4,000,000 and 4,350,000; the hemoglobin was 70 per cent (Tallqvist). The white blood cell count ranged between 6,100 and 9,100 with a normal differential count and smear. Phenolsulphonphthalein excretion following intramuscular injection of the dye after two hours and ten minutes varied from fifty per cent on admission to twenty-five per cent shortly before death. Lumbar puncture showed an initial pressure of 250 mm. of water with a positive globulin test and a total protein of 75 mg. per cent.

X-rays of the skull and kidneys were negative.

Clinical Course: Her course during the remaining forty-nine days of her life was characterized by apathy and listlessness, with frequent headaches and occasional sensations of numbness and tingling in various parts of the body. The blood pressure fluctuated about 200 systolic and 150 diastolic. The eye ground changes were progressively increased in severity. On the forty-ninth day of her stay she suddenly became comatose and shortly thereafter expired.

Autopsy: (No. A32-86) Autopsy was performed by Drs. Krakower and Schlesinger through a limited abdominal incision. There was a large subconjunctival hemorrhage over the left eye. The glands of the axillae and inguinal regions appeared somewhat enlarged as were those of the peritoneal cavity. There was a moderate amount of yellowish fluid in both pleural cavities, particularly the right.

The right kidney weighed 40 grams and the left 140 grams. The right kidney was small. The ureters and vessels were normal in position and origin. The right renal artery was small. The capsule of the right kidney stripped easily revealing moderate fetal lobulations. The surface was otherwise not remarkable. On section through the long axis of the right kidney a sharp line of demarcation between the upper and lower portions was noted 5.5 cm. from the upper pole. The entire kidney measured 10.5 cm. in length. In the upper portion the cortex was well defined and measured 0.2 cm. as compared with 1.3 cm. below that point. The pelvis of the upper portion was somewhat dilated and the medulla thinner than normal indicating the presence of a slight partial hydronephrosis. The cortex in the lower portion of the right kidney appeared swollen and poorly outlined. The pelvis in this portion of the kidney was quite normal. The capsule of the left kidney stripped easily revealing scattered pit-like depressions. The cortex appeared swollen and measured about 1.0 cm. in thickness. The pelvis was not remarkable. Microscopic examination of the atrophic upper portion of the right kidney showed slight cellular proliferation of the glomerular tufts with congestion in some areas but no infiltration. None of the glomeruli appeared hyalinized. The glomerular cap-

sules were thickened, but presented no cellular proliferation. The afferent arterioles showed moderate hyalinization of the media and narrowing of the lumen. The tubules all appeared very small and presented thickening of the basement membrane. The lumina of the tubules were almost entirely obliterated. A slight increase in interstitial fibrous tissue with a few scattered foci of lymphocytic infiltration was noted in the medulla. The larger arteries revealed fibrous thickening of the adventitia and the intima. The microscopic picture in the lower portion of the right kidney and in the entire left kidney was quite different. Here the glomeruli were entirely normal and the afferent arterioles showed no evidence of hyalinization. The convoluted tubules were markedly dilated and in some areas vacuolization of the cells was present. The basement membrane was nowhere thickened. There were a few scarred areas near the cortex, containing a small number of chronic inflammatory cells. There was slight intimal proliferation in the larger arteries.

The heart weighed 240 grams. There was some atheromatous deposit in the aortic valve and in the aortic leaflet of the mitral valve. The coronary vessels were normal except for occasional small atheromatous plaques especially at their origin. The aorta presented a few scattered atheromatous plaques particularly about the orifices of the intercostal and iliac vessels. The elasticity of the aorta was good. The circumference of the aorta in the thoracic region was 4.0 cm. and in the lumbar region 3.0 cm. The gastrointestinal tract was not remarkable except for scattered patches of congestion and pin-point areas of hemorrhage. The adventitia of the colon was thickened. Several of the arteries in it were thrombosed.

The muscularis of the gall bladder was edematous. The adventitia was markedly thickened and fibrosed. The arterioles showed slight hyalinization and marked thickening and fibrosis of the media. Marked thickening of the intima of the arterioles was also noted. Necrosis and thrombosis of the arterioles with hemorrhage and inflammatory cell reaction were present in several areas. A large artery in one section showed marked intimal thickening with thrombosis.

The other organs showed varying degrees of congestion. The arterioles of the intestines, pancreas and adrenals showed slight medial thickening and hyalinization, and those of the spleen presented similar though more marked changes.

Comment: This young woman with a negative past history entered the hospital with a present illness characterized by slight fatigue, occasional frequency and enuresis, slight blurring of vision and hypertension of four months' duration; palpitation of two weeks' duration and severe headache, vomiting and blindness of two days' duration. Physical examination was entirely negative except for hypertension of 200 systolic and 140 diastolic. Ophthalmoscopic examination revealed malignant neuroretinitis. Her course was steadily downhill for the next seven months and was terminated by the sudden onset of coma due apparently to a cerebral accident. Postmortem examination revealed necrosis of the arterioles of the gall bladder. The kidneys were entirely normal except for an early partial hydronephrosis, with resulting atrophy of the upper portion of the right kidney. This patient whose only renal lesion was a partial hydronephrosis of one kidney presented the typical picture of malignant hypertension terminating abruptly with coma.

Case 3. History: P. S. (B. I. H. No. 20013), a fifty-six year old Russian Jewish cattle dealer, with a family history of the death of his father of heart disease and with a past history of typhoid fever in 1919 and a fracture of the right leg in 1913, entered the hospital on October 7, 1933, complaining of vomiting of one day's duration. He was accepted for life insurance in 1928. He was quite well until about a year before admission, when he began to experience severe morning headache and frequent attacks of dizziness and tinnitus. He also became aware of dyspnea and palpitation on exertion, loss of appetite and nocturia of once a night. From time to time, he also had attacks of substernal pain which radiated to the neck and back and occasionally down the left arm. He lost twenty-five pounds in weight in the year preceding admission. Six months before admission all his symptoms became worse and he also developed marked blurring of vision. Several weeks before admission he began to have crampy pains in the hands and feet and the day before admission commenced to vomit.

Physical Examination: The patient was a fairly well-nourished, middle-aged man lying in bed in no distress. The heart was not enlarged. The heart sounds were of good quality. The second aortic sound was accentuated and ringing. The lungs and abdomen were normal. The peripheral vessels were thickened and tortuous. The deep reflexes were somewhat hyperactive and there was a positive Hoffmann's sign on the right. The fingers were slightly clubbed. Blood pressure was 230 systolic and 155 diastolic. Ophthalmoscopic examination revealed pallor of the discs with obliteration of the outlines and peridisc edema. The arterioles were markedly narrowed. There were many areas of hemorrhage and a few patches of exudate.

Laboratory Data: Four urine examinations revealed a specific gravity ranging from 1.004 to 1.015. All the specimens contained very small amounts of albumin and rare erythrocytes and leukocytes as well as hyaline casts on rare occasions. The red blood cell count on admission was 5,390,000 per cu. mm. with a hemoglobin of 90 per cent (Tallqvist). This gradually fell to 3,890,000 per cu. mm. with a hemoglobin of 70 per cent. The white blood cell count fluctuated between 10,200 and 18,800 per cu. mm. with a polymorphonuclear cell count of 85 per cent. The blood smear was normal. The blood Wassermann, Kahn and Hinton reactions were negative. The blood sugar fluctuated between 85 and 101 mg. per cent and the blood nonprotein nitrogen between 27 and 36 mg. per cent. The icteric index was 7.5. Lumbar puncture revealed normal pressure and dynamics, a positive globulin test and a total protein of 110 mg. per cent, sugar of 74 mg. per cent, and chlorides of 618 mg. per cent. The spinal fluid Wassermann was negative. Phenolsulphonphthalein excretion after two hours and ten minutes following intramuscular injection of the dye on two occasions was 15 and 25 per cent respectively.

X-rays of the kidneys showed no abnormalities. A seven-foot film of the heart revealed no cardiac enlargement.

Clinical Course: The patient was given a house diet with fluids to 5000 cc. His blood pressure fluctuated about 230 systolic and 160 diastolic, tending to fall slightly below this level shortly before

death. He became confused soon after admission. Slight improvement followed a venesection of 500 cc. on his fifth day and lumbar puncture on his third and eleventh days. Nevertheless, his mental state became worse and several days before death he became comatose. At this time, signs of pneumonia at the right base were noted. He died on his twenty-first day.

Autopsy: (No. A33-88) Autopsy was performed by Drs. Greenblatt and Schlesinger through a limited abdominal incision two hours postmortem. The right kidney weighed 90 and the left 130 grams. The perirenal fat was adherent to the thickened capsules, but the capsules stripped with ease, presenting only a few points of adhesion. The kidneys, especially the right, were contracted. They presented a finely granular, mottled, yellowish and brownish surface. The cortex of the right kidney was irregular and atrophied, measuring 2 to 5 mm. in thickness. It appeared pale, and yellowish-gray in color. The cortex of the left kidney was similar in appearance but was wider and better defined. The left renal pelvis showed numerous hemorrhagic points. The ureters were normal. Microscopic examination revealed a small amount of patchy scarring. Such areas contained fibrosed glomeruli and small foci of chronic inflammatory cell infiltration. The rest of the glomeruli were swollen and congested. Scattered about were a small number of foci of necrosis. These occurred mainly in the glomeruli, but also in afferent arterioles and small arteries. These areas contained masses of fibrin. A few of the afferent arterioles showed hyalinization and thickening of the media. Most of the arterioles were normal. The larger arteries presented marked intimal proliferation throughout. In some areas, the tubules showed colloid degeneration. In one section, there was a large, old infarct consisting of a mass of edematous scar tissue containing dilated capillaries, a few chronic inflammatory cells, scattered atrophic tubules, and several completely fibrosed glomeruli and tubules. In short, these kidneys showed arteriosclerosis and a small amount of acute necrotizing glomerulitis and arteriolitis.

The heart weighed 425 grams. The left ventricle appeared hypertrophied. The valves and endocardium were normal. The aorta was 6.5 cm. in circumference in the thoracic region, and 7.0 cm. in circumference in the lumbar region. There was atheromatous deposit with ulceration from the arch to the bifurcation, most marked in the lumbar region. The elasticity of the aorta was absent.

The liver presented as incidental findings, two small hemangiomata; there was also congestion and slight central necrosis.

The other organs showed varying degrees of congestion. The arterioles of the intestine and the pancreas presented slight medial thickening and hyalinization while those of the spleen showed more marked changes of the same nature.

Comment: This elderly man with a negative past history entered the hospital with a present illness characterized by morning headaches, dizziness, dyspnea and palpitation on exertion, loss of appetite, nocturia and loss of weight of one year's duration. Physical examination was entirely negative except for a blood pressure of 230 systolic and 155 diastolic, generalized arteriosclerosis and hyperactive tendon reflexes on the right. Ophthalmoscopic examination revealed typical malignant neuroretinitis. An anemia gradually developed while the patient was in the hospital. His renal function showed slight impairment. His course was rapidly downhill and on his twenty-first day he died of bronchopneumonia. Postmortem examination revealed arteriosclerosis and a small amount of necrotizing glomerulitis and arteriolitis of the kidneys. This patient with postmortem findings of a small amount of renal arteriosclerosis and occasional foci of necrosis in the glomeruli and afferent arterioles presented the typical clinical picture of malignant hypertension terminating with bronchopneumonia.

Case 4. History: S. L. (B. I. H. No. 20001), a forty-one year old Russian Jewish clothing worker with a negative family history and with a past history of influenza in 1918, entered the hospital on October 6, 1933, complaining of severe headaches of two years' duration. The patient was quite well until two years before admission, when he began to experience severe headaches, chiefly frontal, lasting eight to twelve hours daily. He also commenced to suffer with nocturia of twice a night and slight palpitation on exertion. He entered a hospital where he was told that his blood pressure was elevated; he was ordered to restrict his meat intake. He was free from headaches until thirteen months before admission when they recurred, lasting each time from one-half to twenty-four hours. They were accompanied by nausea, and rarely by vomiting. He was admitted to another hospital where he was found to have hypertensive changes in the eyegrounds, a diffusely enlarged thyroid gland, sclerosis of the peripheral vessels, an enlarged heart and a blood pressure of 228 systolic and 150 diastolic. All specimens of his urine showed small amounts of albumin and casts. His basal metabolism was plus 25 to 30 per cent. A subtotal thyroidectomy was performed and his blood pressure fell to 170 systolic and 100 diastolic. His basal metabolism at this time was plus four per cent. Microscopic examination of his thyroid gland revealed no evidence of hyperplasia. He was discharged with a diagnosis of essential hypertension and arteriosclerosis. Following discharge, he was well until eight months before admission to the hospital. From this time on he experienced constant recurrences of his headaches, dimness of vision and increased irritability. Six months before admission to this hospital, the patient became depressed and suspicious, and developed delusions. At this time the blood pressure was 208 systolic and 142 diastolic. The patient's headaches and vomiting became worse and were sometimes accompanied by loss of memory and changes in personality. He also had transitory diplopia. His dyspnea, palpitation and weakness increased. In addition, the patient had two attacks of hematuria, each lasting several days; four months and three days respectively before admission to this hospital. He lost 50 pounds in weight in the two years preceding admission.

Physical Examination: The patient was a well-developed, poorly nourished middle-aged man lying in bed in no distress. The heart was not enlarged. The second aortic sound was accentuated. The lungs, abdomen and extremities were normal. The peripheral arteries showed advanced sclerosis. The blood pressure was 230 systolic and 150 diastolic. Ophthalmoscopic examination revealed swelling and hyperemia of both discs and obliteration of the disc margins. The arterioles were narrowed and beaded. There were scattered hemorrhages and patches of exudate.

Laboratory Data: Ten urine examinations showed a specific gravity ranging from 1.008 to 1.020. All the specimens contained albumin in varying quantities. Rare granular casts and leukocytes were seen in the sediments and on one occasion there was a shower of erythrocytes. Four red blood cell counts ranged between 4,660,000 and 5,250,000 per cu. mm. with a hemoglobin of 75 per cent (Tallqvist). The white blood cell count fluctuated between 8,900 and 14,400 per cu. mm. with a normal differential cell count and smear. The blood Wassermann, Kahn and Hinton reactions were negative. The blood sugar was 82 mg. per cent, the icteric index 10, the serum calcium 9.4 mg. per cent and the serum phosphate 4.3 mg. per cent. The blood CO_2 combining power varied between 49 and 54 volumes per cent and the blood nonprotein nitrogen fluctuated between 38 and 51 mg. per cent. Phenolsulphonphthalein excretion two hours and ten minutes following intramuscular injection of the dye varied between five and fifteen per cent on three occasions. Lumbar puncture revealed an initial pressure of 200 mm. of water with normal dynamics, no cells, and negative globulin and Wassermann reactions.

A seven foot heart x-ray revealed no cardiac enlargement.

Electrocardiogram showed normal rhythm, rate 100 to 110, left axis deviation, deep S″ and diphasic T′ and T‴.

Clinical Course: The patient was given a house diet and fluids to 3000 cc. His blood pressure fluctuated about 240 systolic and 160 diastolic. His chief symptom was mental dullness which responded temporarily to venesections and lumbar puncture. There was no great change in his condition and he was discharged on his twenty-seventh day to another hospital where his course continued slowly downhill. He died of bronchopneumonia in December, 1933.

Autopsy: (No. A33-102) Autopsy was performed by Drs. Greenblatt and Schlesinger. Several cc. of clear fluid were found in the peritoneal cavity.

Each kidney weighed 100 grams. The capsules stripped easily, revealing finely granular, mottled, red and gray surfaces. The cortex measured 5 to 7 mm. The right renal pelvis was the site of a large petechial hemorrhage. The left was normal. Microscopic examination revealed moderate congestion and edema, and slight patchy fibrosis with chronic inflammatory cell infiltration. Most of the glomeruli were normal except for slight thickening of the tufts. In rare instances there was thickening of the capsule due to proliferation of the lining cells. The tubules were atrophic, and showed thinning of the lining cells. Many contained precipitated protein and casts of various sorts. The arteries showed moderate intimal thickening. The arterioles in many areas showed intimal proliferation and in rare instances, slight to moderate hyalinization of the media. However, most of the arterioles were free of hyalinization. In short, the kidneys showed a small amount of arteriosclerotic and arteriolosclerotic change.

The heart weighed 400 grams. The coronary arteries were palpable and studded with small atheromatous plaques. The valves and endocardium were normal. The lungs were negative except for generalized congestion and some pneumonia in the left lower lobe.

The aorta showed good elasticity. A moderate amount of patchy atheromatous change was present. The brain weighed 1390 grams. There was marked arteriosclerosis of the superficial arteries. The rest of the organs showed varying degrees of congestion, The arteries of the prostate were slightly sclerosed. The arterioles in the pancreas presented slight medial hyalinization and similar more marked changes were visible in the spleen.

Comment: This middle-aged man with a negative past history entered the hospital with a present illness characterized by severe headaches, loss of weight, nocturia and palpitation of two years' duration, nausea of one year's duration, blurring of vision and mental changes of eight months' duration and two attacks of hematuria shortly before admission. Physical examination was entirely negative except for hypertension of 230 systolic and 150 diastolic, and generalized arteriosclerosis. Ophthalmoscopic examination showed typical malignant neuroretinitis. Laboratory data revealed evidence of slight renal impairment. There was no anemia. Postmortem examination revealed generalized arteriosclerosis. The kidneys showed slight arteriosclerotic and arteriolosclerotic changes. This patient with marked generalized arteriosclerosis and slight arteriosclerotic and arteriolosclerotic nephritis presented the typical picture of malignant hypertension with renal impairment terminating in bronchopneumonia.

Case 5. History: F. C. (B. I. H. No. 23296), a forty-three year old white American garage-man, with a family history of hypertension and vascular disease, and a past history of jaundice fourteen years previously, and of dyspnea on moderate exertion for two years, visited an ophthalmologist on March 30, 1934 complaining of blurring of vision in the right eye of one week's duration. That physician noted the presence of marked retinitis with elevation of the disc four diopters on the right, and marked caliber changes and nicking in the retinal vessels in the left eye. He found the patient's blood pressure to be elevated. The patient was then referred to the Out-Patient Department of the Beth Israel Hospital. He was entirely well until January 1934, when he first noted increasing thirst, nocturia of three to five times a night, temporal headache and attacks of nausea. In March, the headaches became worse, and he suddenly developed the blurring of vision referred to above. He lost eighteen pounds in weight since the onset of the present illness.

Physical examination in the Out-Patient Department was entirely negative except for moderate cardiac enlargement, moderate generalized arteriosclerosis and a blood pressure of 220 systolic and 140 diastolic. Ophthalmoscopic examination revealed hyperemia and edema of the discs, more marked on the right, and peridisc edema. The arterioles showed marked caliber changes and nicking at the arteriovenous crossings. Many fresh and old hemorrhages, as well as patches of exudate, were also noted.

Four urine examinations in the Out-Patient Department revealed a maximum specific gravity of 1.018. A small trace to a trace of albumin was noted in all specimens. The sediment was negative except for occasional leukocytes and rare granular casts. Phenolsulphonphthalein excretion fifteen and thirty minutes following intravenous injection of the dye was 28 and 12 per cent respectively. The red blood cell count was 5,490,000 per cu. mm. with the hemoglobin of 80 per cent (Tallqvist). The white blood cell count ranged between 9,350 and 12,200 per cu. mm. with a normal differential cell count and smear. The blood Kahn and Hinton reactions were nega-

tive. The blood nonprotein nitrogen was 33 mg., and the blood creatinine 1.2 mg. and the blood cholesterol 260 mg. per cent.

The patient was observed repeatedly in the Nephritic Clinic of the Out-Patient Department where gradual loss of weight, fatigability and increasing severity of the headaches were noted. The right optic disc underwent secondary optic atrophy while the left became swollen during this period. The blood pressure showed no significant change. He was admitted to the hospital on July 19, 1934.

Laboratory Data: The red blood cell count was now 4,300,000 per cu. mm. with a hemoglobin of 68 per cent (Tallqvist). The white blood cell count was 9,100 per cu. mm. The blood nonprotein nitrogen was 53 mg. per cent, and the blood creatinine was 2.8 mg. per cent. These values fell to 39 and 1.5 mg. per cent respectively during the next few days. Phenolsulphonphthalein excretion after two hours and ten minutes following intramuscular injection of the dye was 35 per cent. A urine concentration test revealed a maximum specific gravity of 1.020. Lumbar puncture findings were entirely normal except for an initial pressure of 350 mm. of water and a total protein of 40 mg. per cent.

X-rays of the skull and kidneys were negative.

Clinical Course: The patient was given a house diet with fluids to 5000 cc. His blood pressure fluctuated about 200 systolic and 140 diastolic. His headaches improved markedly with complete bed rest and lumbar puncture. His eyegrounds showed no change during the eleven days of his hospital stay. He was discharged to be followed in the Nephritic Clinic of the Out-Patient Department.

His headaches soon recurred, he continued to lose weight and his vision became progressively worse. Magnesium sulphate by mouth relieved the headaches somewhat. The blood pressure fluctuated about 180 systolic and 120 diastolic. Two months after discharge from the hospital, he developed severe persistent projectile vomiting and was therefore readmitted. Physical examination was essentially unchanged. Ophthalmoscopic examination revealed secondary atrophy of the right disc, choking of the left disc, and marked retinitis. The blood pressure was 230 systolic and 160 diastolic.

Laboratory Data: Fifteen urine examinations revealed a maximum specific gravity of 1.012. All the specimens contained a slight trace to a trace of albumin and occasional erythrocytes, leukocytes and granular casts. Five red blood cell counts ranged between 4,000,000 and 4,360,000 per cu. mm. with a hemoglobin of 58 to 60 per cent (Tallqvist). The blood nonprotein nitrogen was 56 mg. per cent on admission and slowly rose to 90 mg. per cent; the blood creatinine rose from 2.8 mg. on admission to 4.9 mg. per cent several days later. Terminally, the blood CO_2 combining power fell from 58.4 to 42.7 volumes per cent. Phenolsulphonphthalein excretion after two hours and ten minutes following intramuscular injection of the dye ranged between five and ten per cent respectively. Lumbar puncture was negative.

Clinical Course: The patient was given a bland diet with fluids to 5000 cc. His blood pressure fell to 210 systolic and 140 diastolic about which level it fluctuated. The vomiting soon ceased but recurred as the blood nonprotein nitrogen rose. The patient became drowsy. During the second week of his stay, the patient commenced to have attacks of paroxysmal dyspnea. He was digitalized and the fluid intake was reduced to 3000 cc. The drowsiness and vomiting became more severe and the patient finally died on the thirty-fourth day of his hospital stay.

Autopsy: (No. A34-109) Autopsy was performed by Drs. Frehling and Schlesinger. The right kidney weighed 100 and the left 90 grams. The capsules were thickened and were stripped from the parenchyma with difficulty. The surfaces thus exposed were uniformly brownish-red in color and appeared coarsely pitted and markedly granular. The cut surfaces revealed the absence of the normal markings. The cortex was poorly defined and measured about 4 mm. in thickness. A moderate increase in the pelvic fat was noted. Microscopic examination revealed marked thickening and hyalinization of the media of the afferent arterioles. Similar changes were noted in the larger arterioles. There was moderate thickening of the intima of the arteries. Many of the glomeruli were fibrosed. All stages of this fibrotic process from slight thickening of the tuft to complete fibrosis and hyalinization were noted. There was a large amount of irregular interstitial fibrosis with chronic inflammatory cell infiltration. The tubules showed a varying amount of degenerative change with formation of casts in some areas. There were, in addition, a small number of foci of acute arteriolitis with thrombosis of the involved vessel and infiltration with acute and chronic inflammatory cells. In some areas, the thrombosed arterioles were completely organized. Similar changes were also observed in a few of the glomeruli and the larger arterioles.

The heart weighed 500 grams and presented marked hypertrophy and dilatation of the left ventricle. The valves were quite normal. Microscopic examination revealed moderate intimal thickening of the small arteries, and a small amount of irregular patchy fibrosis with occasional foci of round cell infiltration. The arterioles were not remarkable. There was a moderate amount of thickening of the intima of the aorta.

The lungs were normal except for slight acute bronchitis and a small amount of patchy peribronchial pneumonia. The liver presented only a small amount of central necrosis due to congestion. The capsule of the spleen was thickened. The interstitial fibrous tissue was also increased. The arterioles of the spleen presented marked hyalin thickening of the media with numerous areas of acute arteriolitis. The pancreatic arterioles presented the same picture though to a lesser degree. There was marked intimal thickening in larger arteries of the pancreas with the formation of fresh thrombi in some areas. The prostate gland, gall bladder and retina presented moderate to marked hyalin thickening of the media of the arterioles and a small amount of acute arteriolitis with occasional thrombosis and hemorrhage. There was also a small amount of arteriolosclerosis in the adrenal glands. In short, there was widespread arteriolosclerosis with marked chronic vascular nephritis and cardiac hypertrophy. There was, in addition, a small amount of acute arteriolitis in the kidneys, pancreas, prostate gland, gall bladder and retina with similar more marked changes in the spleen. A small amount of bronchopneumonia was present.

Comment: This middle-aged man entered the hospital with a past history of dyspnea on moderate exertion for two years and with a present illness characterized by nocturia, headache, loss of weight and attacks of nausea, all of three months' duration, and blurring of vision of one week's duration. Physical examination was negative except for cardiac hypertrophy, generalized arteriosclerosis, and a blood pressure of 220 systolic and 140 diastolic. Ophthalmoscopic examination revealed typical malignant

neuroretinitis. Laboratory studies showed progressively increasing renal impairment. At postmortem, advanced vascular nephritis and a small amount of acute arteriolitis of the kidneys were noted. There was also widespread arteriosclerosis, cardiac hypertrophy and arteriolosclerosis of the parenchymatous organs most of which contained foci of acute arteriolitis. This patient with marked chronic vascular nephritis and a small amount of acute renal arteriolitis, presented the typical picture of malignant hypertension terminating in uremia.

DISCUSSION

Malignant hypertension has heretofore been considered a form of primary hypertension. According to Fishberg[5], all patients with malignant hypertension have a past history of benign, essential hypertension or evidence of its previous existence in the form of cardiac hypertrophy.

We have observed that in addition to patients with malignant *primary* hypertension, there are many others in whom the malignant phase supervenes as the end stage of a previously unrecognized morbid process characterized by *secondary* hypertension.

Thus, the typical clinical picture of malignant hypertension was found in case 1 of our series, but the autopsy revealed chronic glomerular nephritis. Similarly, other authors have pointed out that the same clinical picture may occur with hypertension secondary to acute, subacute or chronic glomerular nephritis[5].

It has been claimed by some authors[5, 7], that malignant hypertension with renal failure and chronic glomerular nephritis with renal failure may be differentiated by the fact that the latter frequently progresses more slowly and may show remissions in its course. However, cases of acute, subacute, or chronic glomerular nephritis may not infrequently have a very explosive termination in renal failure, at the end of a period of relative quiescence with severe hypertension. It is true that some of these cases of glomerular nephritis may be recognized as such because of the antecedent history of an acute attack with edema, hematuria or hypertension, but in many instances of chronic glomerular nephritis it is not possible to obtain such data. These latter cases of chronic glomerular nephritis with secondary hypertension are indistinguishable clinically from cases of primary malignant hypertension. Such patients are usually regarded as having primary malignant hypertension until the autopsy demonstrates otherwise. MacMahon[13] and Klemperer and Otani[14] have made similar observations.

Longcope[15], in 1933 reported the case of a young woman of eighteen with a three months' history of vomiting, headache, clouding of vision, soreness of the back and marked weight loss, and who on physical examination showed severe hypertension, enlargement of the heart and malignant neuroretinitis. She died in uremia and her kidneys showed extreme contraction due to pyelonephritis. The microscopic examination of the kidneys was not described. Longcope states, "It is remarkable to what extent the insufficiency of renal function may advance in many of these patients (with pyelonephritis), without noticeable impairment of the health of the individual. . . . When serious symptoms of renal failure appear, however, the course is often very rapid, and within a period of a few weeks, the patient may die in uremia." Similar cases have been reported by Cain[16], and Mallory[17]. The latter author noted the presence of acute arteriolitis in the kidneys of his patient.

Certain cases of chronic lead poisoning with severe hypertension may exhibit convulsive seizures or other acute cerebral manifestations. Such patients may present the typical neuroretinopathy of malignant hypertension[18].

Oppenheimer and Fishberg[19] in 1924, described a patient who presented a short history of severe hypertension with physical findings of severe diastolic hypertension, malignant neuroretinitis and cardiac hypertrophy, with death from cardiac insufficiency; autopsy revealed hypernephroma of the right suprarenal gland, multiple adenomata of the left suprarenal gland, and anatomically intact kidneys. Winkel's[20] case also falls into this category.

Friedberg and Gross'[21] series of cases of rheumatic heart disease with periarteritis nodosa contain one (case 4) with a history and physical findings typical of malignant hypertension. The kidneys in this case showed necrotizing arteritis. Reports of other similar cases of periarteritis nodosa with or without rheumatic heart disease are to be found in the literature[22, 23, 24].

MacMahon, Close and Haas[25] state that pituitary basophilism may have some features in common with malignant nephrosclerosis.

Klemperer and Otani[14] have commented on the striking similarity between many of the manifestations of hyperemesis gravidarum and malignant hypertension.

It is apparent, therefore, that the clinical picture of malignant hypertension may occur in a wide variety of diseases in which secondary hypertension occurs, and that the presence of these underlying morbid processes may frequently be unsuspected.

In addition to those patients who develop the syndrome of malignant hypertension late in the course of either primary or secondary hypertension, there is another group of patients presenting the same clinical picture in whom

there is absolutely no evidence at necropsy of any long-standing previously existing hypertension. Thus, in case 2 of our series, postmortem examination showed no evidence of cardiac hypertrophy and no widespread arteriosclerosis. It is reasonable to believe that in this case the syndrome arose "de novo".

It may well be that the reason certain pathologists believe malignant hypertension is a specific disease is the fact that they have grouped together series of pathologically identical cases and have found that they presented similar clinical features. If, however, an attempt is made to group together cases which resemble each other clinically, as has been done in this paper, it will be found that such a group of cases presenting the clinical picture of malignant hypertension will show a wide variety of pathological processes associated with primary or secondary hypertension.

A consideration of all of the foregoing data makes it possible to classify all patients presenting the clinical picture of malignant hypertension into the following groups:

1. Primary Hypertension.
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   g. Hyperemesis gravidarum.

The concept that malignant hypertension is a syndrome which may occur in patients with either primary or secondary hypertension explains the wide variety of pathological pictures found in the kidneys of such patients postmortem. The histology of the kidney varies depending upon the duration of the elevation of the blood pressure if it is primary, and with the nature of the underlying morbid process if the hypertension is secondary. In some instances, the kidneys are essentially sound (case 2).

Some authors[1, 2, 3, 5, 14] consider the essential pathology of malignant hypertension to consist in necrotizing renal arteriolitis. It has, however, been shown repeatedly[6, 7, 16] that the kidneys of patients with malignant hypertension may contain no such lesions (case 2) or an extremely small number[7] (also cases 3 and 4 of our series). In such instances foci of acute necrotizing arteriolitis may be more numerous in viscera other than the kidneys (cases 2, 3 and 4), this may also be observed in patients whose kidneys show many such lesions (case 5).

Renal insufficiency was formerly held to be an invariable concomitant of the presence of necrotizing arteriolitis in the kidneys, but this is no longer believed to be true. Murphy and Grill[7] and Klemperer and Otani[14] described such lesions in the kidneys of patients in whom there was not the slightest sign of renal insufficiency. On the other hand, Murphy and Grill[7], Cain[16], and Keith, Wagener, and Kernohan[6], failed to observe renal arteriolar necrosis in a number of instances in which death was definitely due to uremia. Necrotizing arteriolitis has also been frequently found in the kidneys of patients with hypertension secondary to various forms of renal pathology. Such patients may or may not present the typical syndrome of malignant hypertension. Renal necrotizing arteriolitis therefore cannot be considered specific for any single morbid process. Its presence may, however, indicate an unusually severe degree or prolonged duration of hypertension.

The nature of the underlying pathological process responsible for the appearance of the clinical syndrome of malignant hypertension apparently does not influence the course of the syndrome. In all cases the prognosis appears to be uniformly poor.

CONCLUSIONS

1. Malignant hypertension is a syndrome which may occur,
   a. With no evidence of previously existing hypertension.
   b. As the end stage of essential hypertension, with or without uremia.
   c. As the end stage of a miscellaneous group of conditions, characterized by hypertension secondary to acute, subacute or chronic glomerular nephritis, pyelonephritis, adrenal tumor, pituitary basophilism, periarteritis nodosa, hyperemesis gravidarum, chronic lead poisoning, et cetera.
2. It is impossible to decide during the life of a patient exhibiting the syndrome of malignant hypertension whether the hypertension is primary, or secondary to some unrecognized morbid process.
3. Since malignant hypertension is a syndrome and not a specific disease, the renal pathological picture will show wide variation from patient to patient. The presence of acute necrotizing arteriolitis does not establish the diagnosis of primary malignant hypertension, nor does its absence rule it out.
4. Whatever the nature of the underlying pathological process responsible for the appearance of the syndrome of malignant hypertension is, the prognosis is uniformly poor.

neuroretinitis. Laboratory studies showed progressively increasing renal impairment. At postmortem, advanced vascular nephritis and a small amount of acute arteriolitis of the kidneys were noted. There was also widespread arteriosclerosis, cardiac hypertrophy and arteriolosclerosis of the parenchymatous organs most of which contained foci of acute arteriolitis. This patient with marked chronic vascular nephritis and a small amount of acute renal arteriolitis, presented the typical picture of malignant hypertension terminating in uremia.

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The nature of the underlying pathological process responsible for the appearance of the clinical syndrome of malignant hypertension apparently does not influence the course of the syndrome. In all cases the prognosis appears to be uniformly poor.

CONCLUSIONS

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3. Since malignant hypertension is a syndrome and not a specific disease, the renal pathological picture will show wide variation from patient to patient. The presence of acute necrotizing arteriolitis does not establish the diagnosis of primary malignant hypertension, nor does its absence rule it out.
4. Whatever the nature of the underlying pathological process responsible for the appearance of the syndrome of malignant hypertension is, the prognosis is uniformly poor.

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TRAUMATIC RUPTURE OF THE LIVER

BY WILLIAM M. SHEDDEN, M.D.,* AND FULTON JOHNSTON, M.D.*

FROM the time of Hippocrates till the latter part of the nineteenth century wounds of the liver were considered as fatal, although isolated cases of spontaneous recovery from serious hepatic injuries were recorded from time to time. An interesting example of this was reported by Fabricius Hildanus[36] in the early part of the seventeenth century. A young man was stabbed during a quarrel and suffered a severe hemorrhage. A large piece of liver appeared in the wound and was removed by forceps. In spite of this fact the patient recovered.

Although many operations have been performed on the liver and with a steadily decreasing mortality it can still be said that surgery of this organ is an imperfectly developed field. From the beginning hepatic surgery has been feared and shunned because of hemorrhage.

Spontaneous hemostasis of a traumatized liver is rare and for the following reasons:

(a) The thin-walled hepatic veins are without valves and tear easily, gaping without retracting or contracting.
(b) Blood mixed with bile coagulates slowly.
(c) The respiratory movements of the diaphragm and abdominal wall produce a continuous variation of blood pressure.

Tillmanns[85] in 1879, after removing wedge-shaped pieces from the livers of animals, closed the abdominal wound without liver suture. All his animals recovered. He concluded that hepatic wounds are dangerous only when large vessels are involved, and that the low blood pressure and slow current in the liver vessels favor a more rapid hemostasis than in other organs.

Langenbuch[46] in 1888 controlled a severe secondary hemorrhage from the liver with mass ligatures. The patient recovered.

It was discovered during the next decade that animals could survive although large amounts of liver were removed. Ponfick[63] in 1890 found that his experimental animals could live though retaining only one-eighth of their original hepatic tissue. It seemed reasonable, then, that the operator need not fear any marked deleterious effect upon the system from considerable loss of liver substance; and with the control of asepsis, hemorrhage remained as the outstanding danger from hepatic trauma.

Clementi[18] in 1890 first used temporary compression clamps to effect hemostasis. The following year Ullmann[88] employed cautery and the infolding of the wound over a tampon for the same reason.

Kousnetzoff and Penski[43] in 1894 laid great stress upon the use of a supple needle of the type of Hagedorn armed with a thick double thread which was passed back and forth through the liver wound. This needle was blunted so that it would be pushed aside by, rather than transfix, hepatic vessels. He circumstitched the larger vessels in the cut surface and ligated them. The same year while Snegirew[71] was ad-

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vising the use of steam as a hemostatic, Cecherelli[16] and Bianchi were advocating whalebone and later decalcified bone plates on the upper and lower surfaces of the liver with sutures through them, leaving the ends of the sutures long and ligating them over the cut surface. The decalcified bone was usually completely absorbed. Other plates of various sorts have been used: ivory, magnesium and even slices of turnip and potato.

In 1902 Carl Beck[4] slit a broad band from each border of the abdominal wound including peritoneum, fascia and in some cases also muscle and leaving one end attached to the abdomen. These strips were used as supports for suture material. During the same year Kocher[41] employed compression forceps to crush the liver substance. These forceps were removed after twenty-four hours.

In 1905 while Cullen[19] was using overlapping mattress sutures to effect hemostasis, Gillette[31] was passing ligatures through the liver substance, thence through the abdominal wall. These made an exit between the ribs and were tied around the ribs on the cutaneous surface.

Pringle[64] in 1908 employed digital compression of the portal vein at the foramen of Winslow which completely arrested all bleeding from an extensive laceration of the liver.

In 1912 Boljarski[8] recommended an isolated omental plastic with suture, and Halsted drew omentum through a large perforating wound of the liver with an excellent result, as regards hemostasis.

McDill[49] in the same year clamped the vessels in the gastrohepatic omentum with an enterostomy clamp. He stated that procedures can thus "probably with safety be made entirely bloodless for at least 8 to 10 minutes."

From the fact that so many methods and modifications of methods of hemostasis have been advocated and also from the fact that combinations of three or four methods have been resorted to, we must infer that there is no one method which can be relied upon for all occasions.

Though the liver is more frequently injured than any other solid organ, yet rupture of this viscus is one of the rarest reported surgical emergencies. However, it is probably more common than the reported cases would indicate as hepatic lacerations without distinctive symptoms undoubtedly pass undiagnosed and are included under the general caption of shock. It appears approximately once in every thirteen hundred cases brought to the accident room of any large general hospital, yet in a series of 365 cases of subcutaneous injury of solid viscera studied by Edler[25] the liver was injured in over fifty per cent.

The liver's large size, its friability, and above all its fixed position between the unyielding prominent vertebral column posteriorly and the firm costal margin and the abdominal wall anteriorly, all combine to render it particularly vulnerable to external force. The fact that the site of injury is most commonly along the posterior surface where the liver lies in contact with the vertebral column indicates that anteroposterior compression is the commonest cause of laceration.

According to Moynihan[55] the subcutaneous wounds are of three kinds: (1) rupture of liver tissue with laceration of Glisson's capsule, (2) separation of the capsule with subcapsular hemorrhage, (3) central rupture leading to hematoma and thence to abscess or cyst. He further states that the right lobe is injured six times as often as the left. Occasionally cases are first seen at quite an interval after injury. These would obviously be in the groups designated as No. 2 or No. 3.

Robertson and Graham[67] report the case of a boy of fourteen who, three weeks after an automobile accident was found to have a large tumor in the right upper abdomen. He had suffered no undue pain. On incising the liver capsule "two quarts of old blood and bile were evacuated". His convalescence was complicated by a sinus which drained old blood and bile for five months.

A similar case is that of a girl who suffered an abdominal injury while coasting. During the next four weeks she occasionally vomited bright blood and had persistent epistaxis. She developed a marked anemia and a high fever. Autopsy showed an immense cavity in the right lobe of the liver.

If the tumor occurs soon after the injury it can be assumed that the content is blood. If it is late in forming, bile will constitute an important volume of the content of the cavity. Here marsupialization might be employed.

When traumatized the liver has a tendency to split or crack in a stellate manner with massive hemorrhage accompanied by varying amounts of bile. Frequently other abdominal viscera or thoracic organs or the diaphragm are also injured, thus producing more or less serious complications which may mask symptoms referable to the injured liver.

Aside from acute anemia and shock, the high mortality in these cases may be in part caused by gastrointestinal stasis with its resulting toxemia as an effect of the trauma in the peritoneal cavity or leakage of bile. Another factor which keeps the mortality high is an imperfect estimate of the gravity of the situation when the case is first seen; for, occasionally a relatively trivial trauma may cause rupture, or severe hepatic injury may be accompanied by comparatively insignificant primary symptoms. There may be little or no pain or shock at first, and the individual may keep on for several hours with his usual activities without suffering much inconvenience. Robin[66] describes a case of a man who fell across a ditch striking his abdo-

men. He suffered only slight discomfort, walked to a car and was driven home. More than twelve hours later he was found to be bleeding severely from hepatic rupture.

SYMPTOMATOLOGY

The chief clinical features are as follows: (1) Pain in the right upper quadrant, though the pain may be generalized throughout the abdomen or not present at all. McKnight[50] states that, if the convex portion of the right lobe is injured, pain is referred to the right scapular region while if the concave portion is involved the discomfort is referred to the waistline anteriorly. Bloch[7] reports a case of ruptured liver in which the maximum tenderness was in the left lower quadrant. (2) Boardlike hardness and exquisite tenderness over the involved area as a rule. (3) Increase in liver dullness, upward or downward. (4) Shock, due to the single impact or to the flooding of the peritoneal cavity with blood or bile, though there have been many reports of cases of ruptured liver with little or no clinical signs of this phenomenon. Noble reports the case of an individual struck by an automobile who showed at the time of the accident no evidence of shock or, in fact, of any injury at all. Twenty-four hours later laparotomy revealed a rent in the region of the hilus. Death ensued five hours after the operation. The degree of immediate shock, therefore, does not represent the injury often enough to be a guide in cases where no shock is observed. Free exposure and hemostasis at the earliest possible moment is advised. (5) A sharp rise in the leucocyte count and a slower fall in the erythrocyte count and hemoglobin. The leucocytic reaction presents a characteristic curve reaching a height of 150 per cent to 300 per cent within the first ten hours. (6) Jaundice, though this phenomenon seldom appears before the third or fourth day, if at all. McKnight[50] advances the theory that the jaundice may be due to the shattering of Glisson's capsule. He reasons that as a result of this laceration the secretory pressure of the liver is decreased and the bile dammed back and forced into the lymphatic channels. It may, of course, also be due to hepatic sepsis. Andersson[1] reports the case of a laborer who developed jaundice two weeks after an abdominal injury. Exploratory laparotomy nine days later revealed a liver abscess. The hepatic capsule was intact. (7) It must be remembered that liver rupture is sometimes accompanied by a slow pulse. McKnight[50] cites the case of an individual who had a pulse rate of seventy twelve hours after the injury though seven hours later a quart of blood was found in the peritoneal cavity. Bradycardia has been produced in dogs by causing hepatic injury, but only when there was a profuse discharge of blood and bile into the free peritoneal cavity.

Thorotrast (25 per cent thorium dioxide) has been employed as a diagnostic aid but its use apparently is not without danger. Burke and Madigan[14] in 1933 report what is probably the first case of liver rupture in which thorotrast was thus used. They state that the patient died of peritonitis on the sixth day after operation. Stewart[76] et al, describe eight cases of trauma to the liver in which thorotrast was employed. Seven of these died but Stewart declares that "none of these deaths is directly attributable to thorotrast".

The thorotrast was found to be deposited in the endothelial cells of the liver and spleen. Stewart[76] believes that this medium probably remains permanently in the endothelial cells of the liver.

Tripoli[86], on the other hand, injected 25 cc. of this solution into each of fifteen individuals "with no immediate or late untoward results". These cases were not, however, complicated by liver trauma.

DIFFERENTIAL DIAGNOSIS

(1) Simple shock. Hourly blood counts will aid in deciding upon or rejecting this diagnosis. (2) Simple traumatism to abdominal wall. Blood examination will help here also. White[89] has also employed diagnostic aspiration of the peritoneal cavity with a large needle. (3) Splenic Rupture. Though the pain and tenderness are usually in the left upper quadrant of the abdomen, the symptoms here may be identical with hepatic rupture and the lack of localizing signs, of course, does not rule out trauma to the liver.

TREATMENT

When possible, immediate operation is essential. The importance of early recognition and prompt institution of surgical measures can best be emphasized by Thöle's[82] statistics. He demonstrated from a study of 260 cases of ruptured liver that if operation takes place within six hours the mortality is 40 per cent, between seven and twelve hours it is 50 per cent, while between thirteen and twenty-four hours it is 67 per cent. After twenty-four hours the mortality rapidly mounts to 86 per cent, though cases have been operated on with recovery two to thirty days after liver rupture.

If blood cannot be obtained from other sources, autotransfusion of blood may be employed. However, the presence of extravasated bile and the possibility of the presence of the contents of hollow viscera should make this a procedure to be done only after careful abdominal exploration. This technic was advocated by Thies[81] in Germany in 1911 and used by Ogilvie[57] in England in 1922. White[89], in 1920, was probably the first to employ autotransfusion in a case of liver rupture in the

United States. Bloch[7] reports a similar case with recovery in 1930, and Robin[66] one in 1934.

It may be found, on opening the abdomen, that the hemorrhage has ceased and that a careful removal of the blood and an abdominal closure are all that is necessary.

If the bleeding is not severe a gauze pack may be sufficient to check the hemorrhage. The hepatoduodenal ligament may be compressed while the packing is inserted. Graham[32] states that this ligament may be safely compressed for a half hour. It is probably well to remove the pack after forty-eight hours under general anesthesia. Drainage introduces infection and occasionally causes a secondary hemorrhage. Robin[66] describes a case in which the pack was left in for two weeks. A liver abscess resulted. The third week the patient had a series of secondary hemorrhages.

If the bleeding is checked when the pack is removed, a piece of rubber may then be inserted to the level of peritoneum in order to take care of a possible later leakage of bile.

If the pack does not check the hemorrhage, suture of the liver should be attempted, employing if possible a large blunt needle.

If a large amount of liver tissue had been damaged it is probably well to administer glucose freely. It has been clearly demonstrated that hepatectomized animals die because of a glucose deficiency.

PROGNOSIS

This seems to depend on:

(a) The amount and rate of hemorrhage.

(b) The escape of bile into the peritoneal cavity which may be a contributory cause of paralytic ileus.

(c) The amount of destruction of liver tissue.

(d) The presence or absence of injury to structures other than the liver.

An extraordinary instance of multiple injury with recovery is reported by Gemmill and Martin[30]. The patient, a woman of twenty-six, was injured by an automobile. There was evisceration of intestine, severe laceration of the liver and a torn right kidney. The intestine was returned to the abdomen, and the liver and kidney sutured. The patient is said to have had an uneventful recovery in forty-six days.

(e) Careful attention to postoperative complications.

The following are case reports of two instances of ruptured liver:

J. T. Case 1. Aged 20 years, single. Entered October 29, 1933.

History. While playing football, the patient was hit across the abdomen by an opponent. This was the first play of the game. The patient played during the remainder of the half, but sat on the sidelines during the second half. He felt pain in the region of the umbilicus and a desire to urinate, but was unable to do so. After the game was over, he lay down on a bench and had difficulty in getting up again. He then rode home. The pain in the region of the umbilicus persisted. He entered the office of one of us (F. J.), at which time an internal injury was suspected and he was referred to the hospital, walking in, three and a half hours after the injury. His pulse was then 100 and his blood pressure 92/60. During the night his blood pressure and pulse readings were as follows:

10:00 P.M. Blood pressure—98/64 Pulse— 88
1:30 A.M. Blood pressure—78/48 Pulse—104
5:30 A.M. Blood pressure—80/52 Pulse—100

One of us (W. M. S.) was called in consultation at six o'clock the morning following entrance.

Physical Examination. At entrance to the hospital, the patient's skin was very pale—grayish-white. He was apparently in great pain.
Head—skull and scalp negative.
Eyes—pupils equal.
Ears—no bleeding.
Nose—no bleeding.
Mouth—negative.
Neck—negative.
Chest—lungs clear. No râles.
Heart—normal sounds. Good quality. No murmurs.
Abdomen—extremely tender and spastic, particularly over the right upper quadrant.
No tenderness in flanks.
No distention.
Extremities—normal.
Rectal—negative.
Urine—no blood.
Fourteen hours after the accident the leucocyte count was 36,000.

A catheter specimen showed no blood.

The spasticity and the tenderness of the whole abdomen, particularly the right upper quadrant, and the high white count suggested that the boy had a ruptured viscus, probably the liver. Despite the fact that his blood and pulse had remained essentially the same for four hours, one of us (F. J.) noticed that his general appearance was distinctly worse. It was decided, therefore, to operate.

Given 1000 cc. 10 per cent glucose intravenous before the operation.

Operation (15 hours after the accident). (W. M. S.) About one and a half litres of blood in the peritoneal cavity. On the posterior surface of the right lobe of the liver there was a good-sized adherent blood clot. This was not disturbed. Transfused with 500 cc. of whole blood.
Six hours after the operation:
Blood pressure—114/75
Pulse—118

November 1st. Pain in the left arm and up to shoulder which persisted for three or four days. Examination of arm negative.
Blood pressure—130/62.
Pulse—96.
Urine—negative.
Blood count—3,000,000 Hgb.—45

November 1st. 2,500,000 Hgb.—50
2nd. 3,200,000 Hgb.—55
3rd. 3,750,000 Hgb.—55

Uneventful convalescence otherwise. Discharged on the fourteenth day.

When seen one and one-half years after the operation, the patient had no symptoms and had a well-healed wound.

R. J. CASE 2. Female. Admitted April 15, 1934.

History. Patient was thrown out of an automobile when it turned sharply and tipped over. She arrived at the hospital within thirty minutes of the accident.

Physical Examination. At entrance her skin was clammy and her pulse almost imperceptible, its rate being about 110. Her blood pressure registered zero. She was semiconscious and could just describe tender areas of abdomen. There were abrasions on forehead and right arm and a laceration at the lateral canthus of the right eye.

Skull—negative.
Pupillary reactions—normal.
Ears and nose—no bleeding.
Mouth—no injuries.
Chest—lungs clear. No râles.
Heart—very rapid. Sounds normal.
Abdomen—quite rigid, markedly so in the right upper quadrant, where there was much tenderness and spasm.
There was no blood in urine.

The examination of the abdomen suggested a ruptured viscus, probably liver. She was getting rapidly worse. It was felt that a white count this early after injury would not aid in a decision as to therapy, for if it were relatively normal we would disregard it. If it were high we would operate anyway as soon as intravenous fluid and, if necessary, transfusion put her in good condition. Exploratory laparotomy, we felt, was urgent.

She was given 1000 cc. of 10 per cent glucose intravenously after which the blood pressure rose to 90/50. Her pulse was of fair quality during the operation, but rose from 116 to 134.

Operation. (W. M. S.) A tear was found in the postero-inferior surface of the right lobe, and bleeding steadily. This was packed with the gauze ends of two cigarette wicks, which caused adequate hemostasis. She was then transfused with 500 cc. of whole blood.

Forty-eight hours after the operation, the cigarette wicks were taken out and a rubber drain was placed through the peritoneal level.

She was in a precarious condition for the first three days and was given intravenous glucose, hypodermoclyses and rectal fluid.

April 17th. W. C.—19,000 R. C.—2.2 Hgb.—55
19th. W. C.— 7,600 R. C.—2.3 Hgb.—55
21st. R. C.—3.1 Hgb.—55
27th. Clay-colored stool, but none thereafter.
27th. Vaginal bleeding.
29th. Still some vaginal bleeding.
May 1st. Dilatation and curettage for incomplete miscarriage. (F. J.)
16th. W. C.—10,800 R. C.—3.2 Hgb.—55
18th. Transfusion of 500 cc. of whole blood.
23rd. W. C.—7,800 R. C.—3.8 Hgb.—75

Temperature. From the first to the eleventh day—from 100.2° down to 98.6°.

From the eleventh day to the thirtieth day—up to 102°.

From the thirtieth day to the thirty-ninth day—from 102° down to 98.6°.

Bile. Bile from the wound from the first to the twelfth day postoperative.

Vomiting. Vomiting small amount from the time of the operation for thirteen days.

Discharged well on the forty-second day.

When seen one year after the operation, the patient had no symptoms and had a well-healed wound.

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BOSTON DISPENSARY VARICOSE VEIN CLINICS

THE ORGANIZATION OF A VARICOSE VEIN CLINIC

BY HILBERT F. DAY, M.D.*

THE Vein Clinic at the Boston Dispensary has been a success because of its organization and because it has continued uninterruptedly for so long. It was one of the first clinics in Boston to appreciate the advantages of the injection treatment of varicose veins and to start treating patients by this method.

At the Boston Dispensary, we consider that organization depends upon

1. Personnel.
2. Quarters.
3. Equipment.
4. The methods of treatment.
5. Follow-up.

The continuation of the Clinic has been made possible by the sustained interest of the personnel as well as that of the institution. The service rendered its patients has maintained the demand for this form of treatment.

As to *Personnel,* we have found that the best organization is to have one surgeon who is responsible for his associates' and assistants' attendance at the clinics and who himself is in charge of part of the work. His service runs throughout the year and this enables him to see that uniform care is given to the patients and a proper division of work carried out. All of our doctors in charge of the Clinic have been for several years in this work. Only doctors who have received special training are allowed to inject veins. Students, internes or graduate students are not allowed to do so until they have observed the methods for a number of times and then only under strict supervision. No doctor is allowed to be in charge of a clinic who has had less than six months' experience in this type of work. We have found that it requires an even longer time to train a man so that he has proper judgment in deciding the type of treatment to institute, and to make him capable of doing his best work.

Our Vein Clinic has been fortunate in having for a long time the same nurse in attendance, one who understands how to help each man and who knows the patients. We think that such a nurse is essential in the smooth running of the Clinic. A volunteer clerk, also, is quite necessary. The volunteer clerk should have some social training so as to be able to meet the patients pleasantly and to handle them properly. This worker, under direction, attends to the follow-up of patients through correspondence, particularly in relation to the completion of treatments. When a larger problem arises, it is handled by a trained social worker. Problems which come under this heading are transportation, the obtaining of the dressings for home care, aid toward bed rest, and the general social problems of relief.

Equipment: Ordinary tables on which the patient may sit or lie are necessary, also stools of various sizes. A dressing table with one or two shelves for bandages, sheet-wadding and ointment jars should be provided for each cubicle, or the table should be so arranged that it can be easily moved from one cubicle to another. On the top of this table, there should be a sterile sheet and on it a sufficient number of syringes and needles prepared for use. On one shelf of the table there should be a blood pressure apparatus, a stethoscope, tourniquets and sterile gauze. The various solutions used for injections, plus adrenalin (for use in case of an untoward reaction) should be conveniently at hand.

Method of Treatment: All new cases are examined in one day by the head of the Clinic, who then maps out for each patient the necessary medical and laboratory work preliminary to their treatment. They are then referred to the Clinic which is in the charge of a doctor particularly interested in some special form of treatment, such as high ligation, multiple injections, the treatment of ulcers and the treatment of phlebitis.

We believe that regular conferences should be held at which the whole staff gives reports on progress in the various methods used and where new methods of treatment are discussed. As to solutions used, we believe that well tried out solutions should be employed until new ones have proved themselves better. For instance, we at first used salt solution for injections, but later the use of quinine urethane became the rule. Now various other solutions are being tried out. In the case of ulcers, at first a simple supporting bandage was used. Now we are sure that a sponge plus adhesive plaster speeds up the healing of an ulcer.

Follow-up: We have found that it is necessary to keep in touch with our patients because they have a tendency to disappear from the Clinic after their primary symptoms have been relieved and before a complete obliteration of the veins, including the long saphenous in the thigh, has been accomplished.

Finally, I would say that the reasons for the success of this Clinic are the special training of the men who take care of it and the adequate follow-up.

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THE TREATMENT OF THE VARICOSE ULCER*

BY EDWARD T. WHITNEY, M.D.,† AND PETER A. CONSALES, M.D.†

FOR success in the treatment of varicose ulcers, the underlying pathology must be recognized. In the first place, the flow of blood in the saphenous veins is downward instead of upward[1] and this puts such an added burden on the deep veins that a second factor is produced; namely, a *generalized* chronic passive congestion of the lower leg and foot. Early in the disease, no definite signs of this chronic passive congestion are present although the symptoms of fatigue, aching and cramps of the lower legs are probably the result of this latter condition. Later on, however, edema, induration of the tissues and discoloration appear with an increase of the symptoms.

A third factor is a *local* passive congestion due to some trauma, skin infection, phlebitis, cellulitis or lymphangitis. The trauma does not need to be very severe because it requires only the smallest amount of added congestion so to diminish the nutrition of the skin that it breaks down. The two most common skin infections in this location which predispose to ulcer formation are the staphylococcus and the epidermophyton, either singly or in symbiosis[2]. A superficial phlebitis temporarily improves the circulation as it stops the reverse flow of the blood in the saphenous veins but after this infectious thrombus is re-canalized, the resulting situation is even worse than it was before, in that, the vein walls are dilated even further, their tone is diminished even more and the chronic passive congestion becomes even more serious than it was previously.

A fourth underlying factor is the tendency of the skin edges to separate once the continuity of the skin has been broken. This is readily observed when the skin is incised. The skin edges of these ulcers tend to separate constantly and the wear and tear on the epithelial margins must, naturally, be very great.

Treatment should correct all of these conditions. To start with, there is always some skin infection in and around an untreated ulcer and, more likely than not, a fungus is there as well, so that a fungicide, as well as a germicide, is called for and, as we are going to cover the whole ulcer leaving no drainage, a generous amount of this antiseptic is necessary. In the clinic, we use a ten per cent mercurochrome ointment or one per cent gentian violet ointment, while in private practice we employ one of the other mercurials such as merthiolate ointment. The ointment is applied to the ulcer and to the skin surrounding the ulcer for a distance of one to two inches. Over all of this are laid strips of adhesive one inch wide, overlapping. This word "laid" is used advisedly because no tension must be put on the skin.

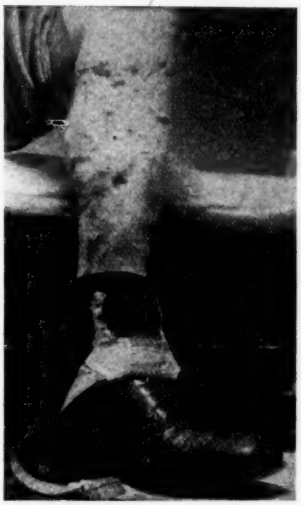

FIG. 1. Mr. S.—Before treatment.

FIG. 2. The left leg is that of Mr. S. after six treatments. The right leg is that of a man treated elsewhere by confinement in bed for eleven weeks. They both had the same sized ulcer, in the same location and with the same number of varicosities. Mr. S. lost no time from work.

The adhesive serves four purposes: First, it keeps the skin edges from separating farther; secondly, it keeps the germicidal ointment in place and prevents it from drying; thirdly, it keeps the ulcer flooded with its own healing secretion and, fourthly, it supplies a base for the application of the sponge which follows. This sponge should be approximately one inch larger

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in diameter than that of the ulcer and should be approximately one inch in thickness. It is laid on the adhesive over the ulcer and then is strapped on with more adhesive, enough pressure being applied to reduce its width by about one-half. McPheeters[3] is responsible for popularizing the sponge treatment in this country, but his directions call for placing fluffy gauze over the ulcer, and in our hands it has been found that such a course in many cases causes gangrene of the skin because too much pressure was produced by the gauze at one particular point. Our modification of strapping the sponge on over adhesive never produces any such gangrene. The object of the sponge is to control the local chronic passive congestion in the base of the ulcer and in the tissues immediately surrounding the ulcer. Furthermore, it presses the newly growing skin edges down into the granulation tissue, providing a thick, well-nourished layer of skin intimately fastened to the underlying tissues rather than a thin, one-celled layer which is easily raised by trauma or infection.

FIG. 3. Ulcer covered with antiseptic ointment.

Injection of the varicosities immediately proximal to the ulcer is made at the first visit and further injection is made at each dressing until all of the varicosities in both the leg and thigh have been obliterated. Personally, we believe in the multiple injections of all of the veins entering the ulcer area with a small amount of sclerosing fluid rather than the injection of a larger amount of fluid at some one point. If it is found that ligation is also indicated, such a procedure can be accomplished at the same time that the ulcer is being treated.

Having dressed the ulcer and having injected the veins, the leg must be bandaged in order to overcome the generalized chronic passive congestion. This can be accomplished in several ways. In the clinic, we use sheet wadding and gauze bandage. Private patients, if they can afford it, may have some form of elastic adhesive bandage. The ordinary ace and Bender bandages do not serve the purpose well enough, in our experience, and elastic or canvas stockings are too expensive to soil. Whatever bandage is chosen should be put on by the doctor treating the patient, as it is almost impossible for the patient to apply a bandage with just the right amount of compression, in fact, a great many doctors require considerable experience before they are able to bandage a leg correctly. Such a bandage is an art, and every one has his own method. Personally, we like to put on a figure of eight going up the leg, and to cover it with spiral coming down. This makes two definite layers of gauze running in opposite directions, and the friction has a tendency to keep the gauze in place. When such a bandage is applied correctly and firmly, the patient will return at the end of a week with the bandage in as good condition as it was when applied.

If the ulcer is large and with an offensive odor, this whole dressing should be changed twice a week and later on every five to seven days. We have found in private practice that at the end of the fifth day the dressing has a tendency to become uncomfortable, the antiseptic ointment has lost its power and without drainage the germs have begun to multiply again, so that the ideal time for changing is approximately just short of a week. In the clinic, it, of necessity, has to be a week. Ether or carbon tetrachloride is used to clean the skin and ulcer at the time of dressing.

Modifications of this treatment must be used for varying conditions. Often the staphylococcus bores little holes in the skin beneath the adhesive beyond the area covered by the antiseptic ointment and when this becomes serious, a good scrub with carbon tetrachloride and an application of a drying powder is applied, covered with gauze only. At the end of a few days, it will be found that this condition has entirely disappeared. Occasionally, patients show idiosyncrasies to various procedures included in the treatment. One here and there cannot stand the sponge, another cannot stand mercurochrome ointment and others cannot stand adhesive plaster. Although many patients state that their skin cannot stand adhesive, it will be found that such statements frequently arise from the fact that staphylococci are growing beneath the skin. A plentiful application of the antiseptic ointment prevents this so that after the first dressing such patients stand the adhesive well enough. Cases which have eczema surrounding the ulcer clear up very nicely following the application of the antiseptic ointment and the adhesive, but in these cases drainage must be allowed at first by not overlapping the adhesive. Many a weeping eczema has been found to have disappeared by the time the ulcer is healed with no other treatment than that given the ulcer.

Occasionally, a large ulcer in the lower part of the leg occurs, with considerable fibrous tissue surrounding it, which does not respond to this type of treatment. Such an ulcer is usually preceded by a deep phlebitis, and the Ho-

mans' operation, or a modification thereof, is about the only method of attack.

The after-care of these ulcer cases is very important. In the first place, after it has entirely healed, the tender skin needs the protection of adhesive for several weeks and this should be changed once a week, never less often than

FIG. 4. Ointment covered with adhesive.

once in ten days, because the skin has a tendency to macerate if the adhesive is left on for a longer period. A very small amount of antiseptic ointment and a layer of adhesive is all that is necessary. However, all the varicosities should be obliterated, and even then the use of a canvas or elastic stocking or bandage should

FIG. 5. Sponge being strapped on.

be continued if there is lymphatic blockage. As we have said before, all legs following deep phlebitis are prone to ulcer formation, probably as a result of the lymphatic system disturbance, so that these swollen postphlebitic legs need careful support for years and maybe for life. This often becomes increasingly important if they show a tendency to ulcer formation. Further protection for the leg can be given, especially in the case of a male patient, by having him wear a leather puttee under his trousers. We have fifteen or twenty men in New England wearing leather puttees in this manner. They are all men who perform some type of manual labor and who have had a recurrence of ulcers due to trauma.

Incidentally, other denudations of the lower leg, not of the varicose variety, such as burns or wide, incised wounds, can be treated to advantage by this method after the active process has quieted down and granulation is beginning to take place.

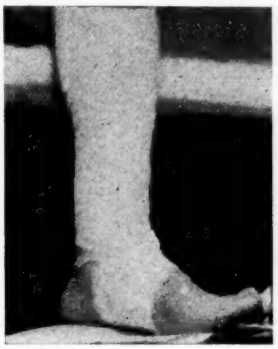

FIG. 6. The whole covered with a supporting bandage.

We see all healed ulcer cases, on an average, every three months, whether they need treatment or not, as many tender pigmented areas have served as forewarnings of trouble, and preventive measures have been instituted early enough to produce successful results.

The length of time required to heal an ulcer definitely depends upon its size, location, chronicity, whether complicated by lymphatic blockage, whether surrounded by much inflammatory fibrosis and induration, and on the amount of local infection in and around it. Those over or distal to the malleoli are particularly stubborn probably because of the wear and tear on the skin edges from the frequent motion of the ankle.

The success of this treatment can be judged from the fact that for nearly three years it has been a standard method in this clinic and in our private work and from the fact that the clinic ulcer cases alone have increased from ten to fifteen a week to fifty to sixty. Such numbers speak for themselves.

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THE TREATMENT OF PHLEBITIS*

BY EDWARD T. WHITNEY, M.D.†

IN order to treat phlebitis properly, it is necessary to recognize the distinction between the phlebitis of non-varicose veins and that of varicose veins.

Phlebitis of the *non-varicose* variety is usually the deep phlebitis of the iliac or femoral veins and sometimes the saphenous. Homans[1] and others[2] have repeatedly shown that not only is the vein damaged but that the lymphatic system surrounding the vein has also been injured. This latter damage, however, results in obstruction or blockade of the lymphatics[2]. The damage is often irreparable and produces a chronic passive congestion of this system. The sign of such a congestion is a brawny edema of the lower leg, either in whole or in certain spots, notably the calf. (Diagram No. 1.)

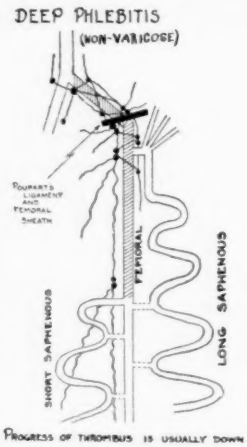

DIAGRAM NO. 1, showing location of a deep phlebitis and the anatomy of related lymphatics. Note how the vessels, including the lymphatic ducts, pass through a bottleneck of fibrous tissue at the femoral sheath. Contracting scar tissue in this area may completely obstruct the lymphatics, especially those in close relationship with the femoral vein from the knee up.

In the vein, a red thrombosis is formed[3]. Such a thrombosis is usually made up of blood elements with very few fibrous elements. The intima may not be entirely destroyed so that the clot may be incased within the vein wall rather than being attached everywhere to the vein wall[3]. Such a thrombosis may extend in the direction of the current as far as the nearest large tributary from which a rapid stream is coming and it may travel distally as far as there is any stagnant blood. It is recanalized fairly quickly in three ways, first, by the natural contraction away from the vein wall of the clot itself[4]; secondly, by solution of the clot by enzymes from the leucocytes within the clot itself[5]; thirdly, by regeneration of any intima that has not been destroyed[3]. The end result in the vein is usually some loss of tone, some possible narrowing and some impairment of the valves.

Treatment must be based upon the principle of helping nature overcome this pathology. This is best accomplished, first, by the application of heat and rest in bed and later by an elastic support for at least several months and perhaps for life. A surprisingly large percentage of our ulcer cases are the result of neglected resolved phlebitis of the deep variety[1, 6]. As a rule, however, this is because of the lymphatic system blockage. Veins are recanalized much more easily than lymphatics are remade or new anastomoses formed. Stagnant lymph lowers skin resistance and allows infection which is ordinarily saprophytic to become parasitic. The result is death of the skin and an ulcer.

In our opinion, if such a phlebitis follows an operation or a delivery, the surgeon or obstetrician should assume the responsibility for it and should supervise after-care as long as it is found to be necessary. So much for the phlebitis of the non-varicose variety.

The phlebitis in a varicose vein presents an entirely different situation. In the first place, embolus rarely occurs for the simple reason that the course of the blood stream is down rather than up[7]. Usually, the phlebitis starts low down in the varicosity and progresses upward, stopping every now and then where the stream becomes somewhat more rapid at the junction of the varicosity and a communicating vein. (Diagram No. 2.) The damage to the lymphatic system is not so important as it is in deep phlebitis because only some of the smaller channels, or ducts, are affected and

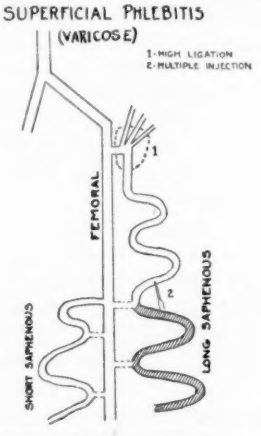

DIAGRAM NO. 2, showing location of varicose vein phlebitis and areas where treatment can be instituted.

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anastomosis between these smaller ducts is more frequent.

Treatment in this type of phlebitis should prevent any extension of the process up the venous system, thereby confining it within the smallest space possible. There are four ways of doing this: first, by excision of the phlebitic vein[1]; secondly, by ligating either well above the affected area or just distal to the femoro-saphenous opening; thirdly, by collapsing the varicosity by very tightly strapping two or three thicknesses of felt padding over the vein wall above the phlebitis; fourthly, by injecting above the affected area[8], thereby producing what Theis[3] calls a white thrombus. Such a thrombus is composed mostly of fibrous or organizing elements and, therefore, is of a more permanent nature than the red or mixed thrombus of the phlebitis. Any one of the latter three obliterations practically immobilizes the phlebitic process and causes it to quiet down within a very few days. The method of choice, where possible, is the injection, as it is of a fairly permanent nature and does not necessitate any operative procedure. These patients formerly were kept in bed for from three to six or seven weeks with an icebag on the affected part, but we know now that this is not necessary, because, aside from the theoretical considerations, a great many of these patients come into the clinic with a phlebitis of many days' or weeks' standing and no harm has come to them.

FIGURE NO. 1. A case of varicose vein phlebitis a few days old. It is being injected in five places just above the proximal border of redness and tenderness. As the difference in color between the phlebitic area and the normal area does not appear in a photograph, a crescentic line has been superimposed indicating the proximal border of the phlebitis.

Having confined or put at rest the phlebitic process, our next step should be to eliminate the underlying pathology which caused the phlebitis. This pathology consists of a chronic passive congestion due to varicosities and a toxic or infectious element. The chronic passive congestion is treated by further injection, ligation if indicated, and support, and the focus of infection, whether local or distant, should be eliminated.

FIGURE NO. 2. Same leg as in figure no. 1. After a lapse of two weeks following injection.

This method of treating phlebitis in varicose veins prevents a three to six weeks' period of total disability, confines the phlebitic or infectious process to the smallest possible space, prevents chronicity or the recurrence of phlebitis after recanalization and prevents future ulceration.

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HIGH LIGATION IN THE TREATMENT OF VARICOSE VEINS*

BY WALTER S. LEVENSON, M.D.†

A FEW years ago the injection treatment of varicose veins became popular in this country. Its popularity was due in a great measure to the fact that a conservative ambulatory procedure was offered in place of a radical surgical operation, necessitating hospitalization and disability. We shared this wave of popularity and enthusiastically entertained visions of relieving a great mass of patients suffering from varicose veins who, for one reason or another, could not absent themselves from their occupations, and who had for years received palliation by numberless procedures. Aside from these considerations, we felt that reports from other clinics here and abroad showed results at least as satisfactory as those to be hoped for by surgery. As time went on, it became quite obvious in our own clinic, as well as in others, that in a certain percentage of cases permanent results were not being obtained, and that patients were returning to the clinic for treatments over a surprisingly long period. Various sclerosing agents and various techniques were used, but it became quite apparent that more radical measures were indicated.

Mindful of the suggestions of De Takats and McPheeters and others, we decided to combine with injection treatment the high ligation of the saphenous vein in selected cases. At the same time, a series of cases was to be treated by the multiple injection method, discussed elsewhere. It was felt that because of the hydrostatic pressure continuously present in the saphenous vein, particularly in the thigh, sometimes injection in those large varices did not work to their best advantage, and that apparently well thrombosed veins recanalized and small branches dilated in from three months to a year.

Therefore, patients with saphenous veins with incompetent valves, but with competent perforating veins, were selected for ligation. New patients and patients who had been previously injected were included.

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†Levenson, Walter S.—Surgeon, Out-Patient Department and Junior Visiting Surgeon, Beth Israel Hospital. For record and address of author see "This Week's Issue," page 991.

TECHNIQUE

Under the usual aseptic technique, infiltration anesthesia with one per cent novocaine solution is obtained. Usually not more than ten to fifteen cc. is necessary. An incision is made about two inches long, two fingers below and parallel to the inguinal ligament with the center over the saphenous vein where it dips through the fossa ovalis. The junction of the saphenous vein and the femoral vein is exposed and the saphenous vein held up by means of a tape moistened in saline solution. Three or four tributaries which are usually constant are isolated and doubly ligated with fine silk and cut between the ligatures. Directly at the junction of the saphenous vein and the femoral vein, the saphenous vein is doubly ligated with number 6 black silk. The saphenous vein is then clamped across distal to these ligatures and transected between the ligatures and the clamp. At this point, the distal segment of the saphenous vein can be injected with ten to thirty cc. of a mixture of dextrose and sodium chloride. After the injection, the saphenous vein is ligated with silk below the clamp and a segment of vein about one-half inch long is removed. The subcutaneous structures are approximated with fine, plain catgut and the skin with interrupted dermol or clips. A collodium dressing is applied and compressed with adhesive plaster and flat gauze sponges. In twenty-four hours the patient is allowed up out of bed and usually goes home in about four to five hours after that, making a total hospital stay after operation of about thirty hours. Ace bandages are applied to the extremity, and the patient is seen again in the clinic in about three days.

Surprisingly little pain is complained of by these patients postoperatively, if no injection is made at the time of operation. At most, only moderate pain is noted if injection of the lower segment is carried out as described above. It is satisfying to observe that even after this operative procedure has been carried out without injection of the saphenous vein in the thigh, a vein which previous to operation was easily palpable is difficult to find, indicating that the back pressure has been controlled. About one week after operation, injections are started again according to our accepted techniques.

THE MULTIPLE INJECTION METHOD OF TREATING VARICOSE VEINS*

BY S. SEYMOUR HORLICK, M.D.†

BY the multiple injection method we refer to the procedure described by McPheeters[1] in his book "Varicose Veins." This method has as its basis the injection of a sclerosing solution into many points along the course of varicose veins, at a single sitting, to produce massive obliteration of their lumina. The advantages in using this method are primarily to reduce time over which treatment is necessary and, secondarily, to make for more efficient obliteration of these pathologic veins.

Statistical data as to the length of time over which treatment is given are lacking in the literature of varicose veins. Faxon's[2] carefully prepared report of 314 cases at the Massachusetts General Hospital, shows an average of about seven injections per patient, with two or three injections per visit. This particular report shows sixty-three per cent recurrence. From our experience, the number of injections would account in good part for the large percentage of recurrence. It is almost inconceivable that one can obliterate the entire system of varicosed veins with an average of seven injections.

From our observation of the other clinics in Boston, we find that the patient makes periodic visits spaced from one to four weeks and, usually, then receives only a single injection and that into the most prominent, or most easily found, varicose vein. At the next visit, the treatment may have no relation to the preceding one and this haphazard method is pursued until the patient no longer returns because he is either tired of so many injections or satisfied since the most noticeable veins have disappeared. We found certain patients who had returned at intervals for five years and were still being treated. This makes us feel that a definite plan of procedure should be mapped out at the first visit and carried out to its completion as promptly as possible.

Theoretically, there is only one absolute contraindication to the injection treatment. That is inadequacy of the deep circulation. There are, however, more contraindications to the use of multiple injections. From a routine examination, one may find that the patient has diabetes, in which case sugar solutions should not be used. The patient may have nephritis, in which case concentrated salines should not be injected in large amounts. Patients with heart disease are often benefited by obliteration of their varicose veins but as a rule should not be subjected to multiple injections because the treatment is a long one, one-half hour or more, and may tire them excessively. The treatment is further contraindicated in neurotic patients because of cramps and venitis, which is often incident to good obliteration. Also in cases of *active phlebitis* of infectious origin. It has aptly been stated[3], "Never treat any patient with varicose veins by the injection method so radically, completely or extensively that she will be incapacitated or bedridden, for, in so doing, one great advantage, that of being an ambulatory treatment, is lost."

Given a patient suitable for multiple injections, the technique is not difficult, but it requires considerable attention to details. An assistant greatly facilitates the work. The time consumed at each visit is necessarily long, but the results compensate for it.

Our technique is that described by McPheeters[1] except for a few minor variations. Briefly it is as follows: The patient is placed in an erect position and the points where injections are to be made are marked with mercurochrome solution or tincture of iodine so that one may know where to inject when the veins are subsequently collapsed. The patient lies down and the extremity to be treated is elevated and stripped of blood. Two tourniquets are applied about six to eight inches apart, starting at the foot or ankle. These must not be applied so tightly as to cut off the deep circulation, for serious trouble might ensue from the sclerosing solution finding its way to a corresponding segment of the deep circulation. After this, the leg is lowered and the points previously marked are injected between the tourniquets with the solution to be used. Pads of gauze are strapped over each puncture wound. This done, the distal of the two tourniquets is removed and the leg again elevated and stripped of blood. The tourniquet previously removed is then applied about six to eight inches above the one left in place and the leg lowered. This segment is then injected as was the lower one. This procedure is followed up the leg and thigh until the veins are injected to the groin. The most proximal of the tourniquets is left in place and the whole extremity is firmly and evenly bandaged from toes to groin. The ace or elastoplast bandage is ideal for this purpose. In our clinic, because the cost of these latter is prohibitive, we use sheet wadding and ordinary bandage with satisfactory results. When the bandage is applied the tourniquet is removed. By this means the veins have been injected while collapsed and have been kept collapsed, as near-

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ly as possible, by the bandage. The resulting thrombi will thus be smaller in diameter. It is advisable to keep the bandage in place until the next visit to the clinic. At the ensuing visit, small varicosed veins, so-called "pick-ups", missed at first, may be individually injected without the use of tourniquets. If the veins of both legs were varicosed and only one done at the first visit, the other may be done at the same time as the "pick-ups".

It is important to obliterate the internal saphenous vein thoroughly. This may frequently be found, when not palpable or seen, by the technique described by McPheeters[4], the so-called "percussion pulse transmitted". This is obtained by percussing a distended loop in the calf or lower thigh with one hand while the other is held flat over the location of the internal saphenous vein. A distinct fluid wave is produced within the vein which is felt by the palpating hand.

A vein, size 3* or 4* in McPheeters' classification, when treated by single injection while distended, will more readily recanalize than the same vein collapsed. In general, it may be stated that the greater the diameter and the shorter the length of the thrombus, the greater will be the probability of recanalization. This is due to pressure created by the weight of a column of blood above the thrombus. If the whole column of blood was thrombosed at the same time in a collapsed system of veins, recanalization is lessened because the thrombus would be narrower, more extensive and, therefore, more resistant to the back pressure.

The patients chosen for multiple injections in our clinic have been those with extensive varicose veins, who showed no signs of heart or kidney disease, active phlebitis or neurosis. Many of those who were barred may well have been treated had it not been for the limitations of facilities for handling large numbers of long treatment cases.

The solutions used in our series of cases were twenty per cent sodium chloride, five per cent sodium morrhuate and seventy-five per cent invert sugar. Needless to say, sclerosing solutions that are toxic in moderate or large dosage, such as quinine hydrochloride and urethane, or the salicylates, must not be used. Quinine hydrochloride and urethane has been used for injecting the "pick-up" veins missed at the multiple treatment. We found that ten cc. of sodium morrhuate in one fourth to one half cc. dosage at each point is suitable for the average case. In more severe cases we supplement this with twenty per cent sodium chloride solution. The latter solution gives an excellent firm thrombus but, because of the severe cramps it causes, we prefer the sodium morrhuate. We have not seen toxic manifestations from this large amount of the morrhuate solution. None of these solutions will produce a slough if the technique is properly executed; and *any of them may produce a slough* if the solution finds its way outside the vein lumen.

One point not sufficiently recognized should here be emphasized. The destructive action of the sclerosing solution on the vein wall continues as long as the solution is in contact with it. The longer the action, the greater the destruction. Hence, it is important to keep the solution localized in a segment of vein just long enough to produce damage to the intima and not erosion through it to destroy the thinned-out media. It is in these cases that one sees severe perivenitis and slough, even though the surgeon was certain he was within the vein.

It is gratifying to note that patients who receive multiple injections suffered less post-injection discomfort. This is probably due to the support afforded by the bandage. Cosmetically the results are superior to those produced by the single injection method.

In an attempt to compare the duration of time consumed in treating patients by the single and multiple injection method, fifty cases of the former, picked at random from the records of the vein clinic of the Boston Dispensary, have been classified. Those records which showed the patients to have advanced myocardial disease, nephritis in any form, neurosis or active phlebitis were excluded. Fifty-one consecutive cases, treated both privately and in the Boston Dispensary Clinic by the multiple injection method, were similarly classified. The private cases were treated by the same technique, with the same solutions and by the same operator as were the clinic patients. Only those records were analyzed which showed that the patient was discharged except for follow-up.

From this analysis, the average patient by the single method received 19.52 injections at 21.76 visits, covering a span of 82.22 weeks. By the multiple injection method, it was found that the patient did receive an average of 44.11 injections at 4.76 visits, covering 4.17 weeks.

In this series of fifty-one cases treated by multiple injections, there were four recurrences and only in those who showed a double Trendelenburg. Patient No. 5 of the Multiple Schedule showed slight recurrence in twelve months. Patient No. 8 showed recurrence in sixteen months. Patient No. 25 showed slight recurrence after twelve months. Patient No. 51 showed slight recurrence after eight months. Excluding three patients who had not received follow-up examinations, the recurrence rate is 8.33 per cent.

*McPheeters' Classification of Veins:
Size 1. ½ centimeter in diameter.
Size 2. ½ to 1 centimeter in diameter.
Size 3. 1 to 1½ centimeters in diameter.
Size 4. 1½ to 2 centimeters in diameter.

SINGLE INJECTION SCHEDULE

Case No.	Age	Sex	No. Injections	No. Visits	Duration in Weeks
1.	47	F	10	10	28
2.	37	F	15	15	32
3.	52	F	8	8	40
4.	36	F	24	28	42
5.	40	M	7	8	9
6.	44	F	23	24	55
7.	47	F	14	14	68
8.	49	F	9	10	28
9.	43	F	27	27	100
10.	47	M	10	11	60
11.	34	F	27	27	104
12.	52	M	35	36	150
13.	44	F	8	8	20
14.	37	F	18	19	64
15.	56	M	48	55	160
16.	45	F	93	128	156
17.	45	F	46	46	154
18.	26	F	27	28	170
19.	37	F	33	38	162
20.	54	F	5	7	40
21.	62	M	20	29	144
22.	54	F	30	38	234
23.	46	M	5	6	88
24.	31	F	25	28	172
25.	34	F	3	4	156
26.	30	F	9	10	162
27.	53	F	4	4	104
28.	47	F	15	15	56
29.	50	F	5	5	36
30.	60	M	12	12	44
31.	39	F	5	5	36
32.	74	F	2	2	12
33.	68	F	4	4	14
34.	50	F	10	10	36
35.	35	F	3	3	15
36.	44	F	30	39	230
37.	39	F	13	15	44
38.	50	F	23	24	40
39.	64	F	16	16	54
40.	24	F	12	12	52
41.	16	M	4	4	24
42.	30	F	14	14	42
43.	39	F	10	10	76
44.	48	F	118	127	224
45.	64	F	12	12	34
46.	45	M	16	16	74
47.	44	F	30	38	100
48.	50	M	10	10	42
49.	60	F	15	15	50
50.	54	M	14	14	74
Averages:	45.52	M 22% F 78%	19.52	21.76	82.22

MULTIPLE INJECTION SCHEDULE

Case No.	Age	Sex	No. Injections	No. Visits	Duration in Weeks
1.	54	F	48	4	3
2.	50	M	56	5	3
3.	43	F	26	2	1
4.	25	F	35	3	3
5.	36	F	64	5	4
6.	29	M	32	2	1
7.	64	F	81	7	8
8.	51	F	35	3	4
9.	25	F	20	2	2
10.	49	M	51	4	3
11.	47	F	47	6	4
12.	30	F	24	2	1
13.	69	F	134	11	8
14.	42	M	50	6	4
15.	51	F	58	5	4
16.	64	F	44	4	4
17.	77	M	43	3	2
18.	28	F	66	7	5
19.	37	F	81	10	12
20.	64	F	36	3	2
21.	51	F	30	4	3
22.	57	F	36	4	3
23.	61	M	44	6	6
24.	43	M	24	2	1
25.	63	F	44	6	4
26.	47	F	46	6	4
27.	54	M	34	3	2
28.	39	F	28	2	1
29.	56	F	54	4	3
30.	54	F	61	5	6
31.	52	F	44	4	4
32.	64	F	35	3	2
33.	62	F	70	6	5
34.	58	F	40	4	5
35.	35	F	44	5	4
36.	56	F	46	5	4
37.	20	M	22	2	1
38.	45	F	70	6	7
39.	58	M	54	5	4
40.	53	F	34	3	3
41.	62	F	30	3	2
42.	80	F	21	2	1
43.	50	F	40	4	4
44.	69	F	66	8	8
45.	52	F	52	9	11
46.	67	M	29	4	3
47.	38	F	23	3	4
48.	40	F	24	8	9
49.	50	F	15	8	8
50.	48	F	28	6	5
51.	55	F	31	9	8
Averages:	50.47	M 21.56% F 78.44%	44.11	4.76	4.17

It should be remembered that the cases here reported were a selected group. They were, in the main part, advanced cases of varicose veins. We should like to examine and report the rate of recurrence in these same patients three to five years hence. The results obtained up to the present, however, are extremely gratifying.

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NEW ENGLAND SURGICAL SOCIETY

PERSONAL EXPERIENCES WITH TUMOR OF THE BLADDER*

BY J. DELLINGER BARNEY, M.D.†

THE literature of tumor of the bladder is already extensive and is growing rapidly. Many studies have been made of large series of cases, the largest and most recent being that collected and analyzed by the Carcinoma Registry of the American Urological Association (*J. Urol.* 31:423 [April] 1934). The conclusion of most urologists of experience is essentially the same, namely that recurrence, no matter what the treatment, is very frequent, that five-year cures are relatively few in number (15 to 30 per cent), and that there is a high operative mortality. A further point of agreement, perhaps one of the most important, is that it is uncommon to see a case in its earliest and therefore most favorable stage.

In view of all this I do not feel that what I shall have to say will contribute anything new or strikingly different from what has already been reported. I do feel, however, that an experience of about twenty-five years with a series of some 250 cases enables me to express an opinion on certain aspects of the question.

Although this is to be by no means a statistical study, a few figures must be given to impress certain old truths upon you. That tumors of the bladder are seen infrequently even by the urologist is shown by the fact that they comprise only about three per cent of all urological cases. Most are seen first by the family doctor, by the internist, or by the general surgeon. I have already stated that it is common experience to find many of these patients with well-advanced cancer before they are finally seen. It is important, therefore, to consider the possibility of improving on this situation. It is not unfair to say that the blame for delay rests partly on the doctor and partly on the patient. To anticipate a little let me say that all studies of such cases show that hematuria, gross or microscopic, intermittent or constant, is the initial and presenting symptom in about eighty per cent. It is also important to know that in various studies of cases of hematuria as a symptom, malignant disease of some part of the urinary tract is responsible in about seventy-five per cent. It follows not only that we must be constantly on the watch for blood in the urine, but also that we must advise the patient to have the source of bleeding investigated. In the event of microscopic blood, involving as it does for its discovery a careful microscopic examination of the sediment, it is not enough to attribute it to a "cold" or a "strain" or some such unlikely cause. We must bear in mind its possible significance, thereby giving the patient the unquestionable advantage of early interference with a situation fraught with disastrous possibilities. Too often are all concerned lulled into a sense of false security by the fact that the bleeding stops after the administration of medicine, or by a period of rest. Experience shows clearly that the bleeding from bladder tumor is very frequently intermittent, and will let up spontaneously for a greater or less length of time. Not only this but there is no drug yet discovered which will influence hematuria favorably. The physician however is handicapped by the fact that the patient may not realize that there is blood in the urine, even sometimes in considerable quantity, therefore and quite naturally not seeking advice. It is also a fact that human nature being what it is, many patients shrink from consulting the doctor because of their fear of being hurt or of being told some unpleasant news. The situation is difficult, and the only solution I can suggest is (a) more widespread use of the annual or semi-annual routine physical examination, and (b) more frequent and more forceful spread of the facts before both the profession and the laity.

Many other writers have shown the apparently unnecessary delay in making the diagnosis in tumors of the bladder. In my own cases sixty-six patients said they had had symptoms, consisting not alone of hematuria, but also of dysuria, frequency, loss of weight, et cetera, lasting anywhere from one to twenty years; in a smaller number symptoms had been noted for many months. In a series of 902 cases reviewed by the Carcinoma Registry of the American Urological Association, a diagnosis based on complete examination was made within one month from the beginning of symptoms in only 10.8 per cent, while in 48.3 per cent the diagnosis was delayed for more than a year. Such a situation would appear to be wholly unnecessary in view of the diagnostic and therapeutic attainments of today. Let us all work together for its improvement.

But there are other symptoms, almost as common, which, together with hematuria, characterize tumor of the bladder. I refer to frequency of urination, occurring in over seventy per cent of my cases, and dysuria, found in over

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forty per cent. We have, therefore, a very definite diagnostic triad, with hematuria forming the largest leg, on which to base a probable diagnosis, and one which can be confirmed sometimes by abdominal examination, more often by rectal or vaginal examination, and always by cystoscopic study. As a matter of fact in my own series of 250 cases, rectal examination in the male was suggestive or definitely indicative of tumor by way of induration, nodularity or thickening in forty-six per cent; vaginal examination yielded similarly positive findings in forty-four per cent. In this connection it is also desirable to emphasize the importance of bimanual examination, either rectal or vaginal, as in twenty-nine per cent of my cases this showed evidences of a mass in the bladder.

In the series of 902 cases already referred to loss of weight was noted in 251 or 27.8 per cent, and gastro-intestinal symptoms in 128 or 14.1 per cent.

The hematuria, gross or microscopic, of bladder tumor is frequently referred to as a "painless hematuria". And so it is in many instances, especially in the early stages of the disease. When found it is almost pathognomonic. But we have also seen that it is accompanied in many cases by other significant symptoms, the natural history of which I would like to dwell upon for a moment. It is easy to see how such a highly vascular structure, often rapidly growing, as a bladder tumor should bleed. Frequency and dysuria are the natural sequences, being induced by necrosis of the growth with subsequent infection, which involves not only the neoplasm but the bladder as well. This condition is shown by the fact that not only blood but also pus and albumin are found in more than half of any group of cases.

That the bladder symptoms are probably induced not only by infection, but also by the position of the tumor is demonstrated by the fact that these tumors originate in the trigonum in from one-quarter to one-third of the cases, and on the lateral walls and bladder neck in from one-third to one-half of the cases. That the position of the tumor near or actually within what one might call in rough language the "business end" of the bladder is an important contributing factor to the disturbance of urinary function, is shown by the fact that in those rather infrequent instances where the neoplasm is in the vault of the bladder (about 7.5 per cent), urinary symptoms, even hematuria, are far less conspicuous until the disease is well advanced.

From what has gone before it is now possible to see why we find loss of weight and gastrointestinal disturbances so frequent a factor in these cases. Long-continued hematuria, interspersed with exacerbations of sometimes marked severity, together with infection, are factors sufficiently important. But in addition to this we have another element and that is kidney damage. That this is not infrequent has long been recognized, more especially in recent years, since the advent of intravenous pyelography. By this method of investigation one can readily establish not only its presence, but also its extent, and whether one or both kidneys share in the destructive process. Not only is renal involvement demonstrated in this way, but it is also shown by the fact that the nonprotein nitrogen or urea nitrogen figure is abnormally high in the great majority of cases. In the 902 cases already mentioned the blood chemistry was investigated in 546. In 472 the results were indicative of serious kidney damage, sometimes excessive. Further evidence of renal impairment is borne out by autopsy observations. In the series mentioned there were seventy-nine postmortems of which twenty-six showed infection or dilatation or both of one or both kidneys and ureters. As additional evidence I cite the statement by Smith and Mintz (*Am. J. Surg.* 20:55 [April] 1933) that in "36 cases in which the condition of the kidneys was shown at autopsy, both kidneys were normal in but nine".

Various factors contribute to this destruction of the upper urinary tract. That actual obstruction of the ureter by the pressure or actual invasion of the growth is a factor is shown by the fact that it was found in eleven of the seventy-nine autopsies referred to. Where actual mechanical obstruction cannot be found the changes may well be induced by severe infection, by obstructing lesions at the bladder neck, and by cicatrization, both of the latter conditions sometimes resulting from the operation.

Here therefore is a partial explanation of a high operative mortality, thirty per cent or more. Other factors in the order of their frequency are circulatory accidents, pneumonia, peritonitis, shock and sepsis. Before proceeding to the next phase of this problem I can only emphasize again the enormous importance of an early diagnosis if we are to enable the patient to escape the disasters which are bound to overtake him if he waits long enough. Delay in the recognition of these tumors is a prime factor in this very high operative mortality.

Throughout my remarks I have referred to "tumors" of the bladder, meaning by this carcinoma. I think it is generally accepted that all epithelial tumors of the bladder are at least potentially malignant, regardless of their apparently benign appearance under the microscope. Although these are classified as Grade I, II, III, or IV according to their content of differentiated and undifferentiated epithelium, experience shows that the grade of malignancy by this classification seems to bear no definite relation to their tendency to metastasize. It seems to be a fact, however, that cancer of the vault of the

bladder is generally of a higher grade of malignancy than that elsewhere. For this reason and because through lack of symptoms they are apt to be well advanced when discovered, the prognosis is less favorable than for those in other parts of the bladder. It also seems to be a fact that the papillary type of carcinoma is far less likely to form metastases than the squamous type. Furthermore experience shows that while "less than half of the tumors" in the trigone, bladder neck and lateral walls are "highly malignant", seventy-five per cent of those in the vault show the higher grades of malignancy. Still another point to be emphasized is that while about one-third of all bladder tumors are multiple and that only about one out of three of these is highly malignant, almost one-half of the single tumors (these constituting about two-thirds of any given series) are of grades III or IV.

In this connection and before proceeding to other phases of this important subject, it is worthy of note that in the series of 902 cases studied by the Carcinoma Registry nearly half of the tumors had attained a diameter of more than 5 cm. when they were first observed. Here again is an indication of delay in their recognition, for to reach such a size the tumor must have existed over a considerable period of time. This, together with the fact that experience has shown that the results in tumors of less than 2 cm. are much more favorable, offers still another argument for prompt diagnosis and early intervention.

Although it has generally been believed that bladder tumors metastasize infrequently or only late in the disease, autopsy observations and recent intensive studies by various authorities show that metastases occur in at least ten per cent of cases. In a series of thirty-four autopsies reported by Smith and Mintz, metastases were found sixteen times (47 per cent). We do not yet know the entire story, for even the relatively few who come to autopsy can be so thoroughly examined as to be sure that no metastases exist. These deposits are found far more frequently in the bones than elsewhere, with the lungs, regional lymph nodes, liver, retroperitoneal nodes and peritoneum next in order of frequency. Nor is it impossible to exclude metastases, even though the tumor of the bladder has at least been temporarily eradicated. A patient of mine, a physician, died of metastatic cancer of the brain some months after his third operation for cancer of the bladder and when a recent cystoscopy showed no evidence of recurrence.

Treatment of cancer of the bladder resolves itself into five categories. A very few small lesions, sometimes accidentally discovered, of the pedunculated and so-called benign epithelial group can be cured by fulguration through the cystoscope. These may be or may become multiple. I have had several of these definitely cured as shown by the elapse of more than five years without recurrence. Only recently however I saw a woman whose tumor I fulgurated five years ago, and who now, after complete freedom from neoplasm, has developed two small growths similar to those first seen.

In very unusual instances a sessile or infiltrating growth of small size (not over 1 cm. in diameter) and in a readily accessible position may be cured by the cystoscopic implantation into its base of one, two or three radon seeds. Although I have had a few such cases result favorably to date, I feel it is justifiable to advise an operation as offering greater chance of permanent cure.

The external application of roentgen rays has been tried in certain cases both as a therapeutic measure and as a preoperative procedure with the idea that it will retard growth and by the production of a fibrosis make metastases at least by way of the lymph channels less likely. While the last word has not yet been said on this method of treatment, I think it is of little value and should be abandoned unless as a last and sole resort. It is, however, of considerable value in relieving the distressing cystitis of advanced cases, and the pain produced by metastases.

Wide resection of the growth with ample margins of healthy tissue is, when possible, the best method of removal. It does, however, result in a mortality of about thirty per cent and cannot be applied in those cases where the tumor is located in the trigone, bladder neck or low lateral wall.

Fulguration, by which I mean removal of the growth with the cautery loop, with or without the implantation of radium seeds in the base, carried a somewhat lower mortality but gives no greater prospect of cure than resection. This method of treatment is peculiarly suited to those tumors which are found in situations not suited for wide resection and which we have seen form the bulk of the cases.

The highest mortality (about 40 per cent) seems to occur where chief reliance is placed on the implantation of radium seeds in large numbers and where only that portion of the tumor which protrudes above the level of the bladder mucosa can be removed by cautery loop or knife. Here we have various factors contributing to the mortality among which toxemia both from radium necrosis and from sepsis are the most important. Not only this but even if the patient does not die, he may be made wretched by the severity and long duration of the radium burns.

As a matter of fact the final results do not seem to be better by one method of treatment than another, and in approaching a case the surgeon must be prepared to make use of any

or all of the procedures touched upon. When we realize that a recurrence of nearly fifty per cent takes place after any form of treatment, that only about one-third of the cases treated survive for five years or more, and that there is an operative mortality of over thirty per cent, we realize that a very real problem confronts us. Because of this we must give it our most serious thought. Not infrequently the most unfavorable sort of a case will turn out most satisfactorily; on the other hand what seems to be a more easily controlled tumor will prove to be of the most vicious variety.

Before closing I wish to say a word about total cystectomy which, theoretically speaking, should offer a satisfactory solution of our problem. But like all other apparently good things it has its drawbacks. Even in the supposedly favorable case there may be undemonstrable metastases. Then, too, when we say cystectomy we mean also one or two additional preliminary and major operations for the diversion of the urinary stream. Having survived these ordeals removal of the bladder itself may tip the scales, coming as it does in the footsteps of other surgical procedures of real magnitude. In spite of this I think we are all convinced that it offers the only definite hope of cure in certain carefully selected cases. After all, the patient will surely die if he is not cured of cancer, and in the process will drag out a most wretched existence. Should we not therefore give him all the chance that surgery offers to obtain definite and permanent relief?

The length of time required and difficulties encountered in the follow-up of patients, whether private or hospital cases, is well known. While this undertaking has been begun it is by no means complete. Not only this but it involves a good deal more than a mere statement as to how long a patient lived after operation. This aspect of the subject will therefore be dealt with at a later time in another communication.

NEW HAMPSHIRE MEDICAL SOCIETY

PRESIDENT'S ADDRESS*

BY FREDERIC P. LORD, M.D.†

ONE of the temptations of an occasion like this, where opportunity is given me, or is forced upon me, to talk before the members of this Society with no discussion to follow, is to get rid of any wild and pet ideas which I may be harboring. There is no lack of such, of course, on my part, but I have decided to resist this special manner of suicide.

The logical procedure would be for me to talk about the one thing concerning which I may be supposed to speak with some authority. As I thought of you listening to a long paper on some anatomical subject, in which I should be enthusiastically interested, I tried to picture your state of mind, feeling you were brought back again to your medical school days. I feared I should need to ask to have the doors locked before I could finish the paper.

Then there is the perennial subject, a vitally important one, to which you have frequently listened at this annual meeting and elsewhere, The Socialization of Medicine. Certainly I have my own ideas on the subject, and here is a chance to recount them. But can I give you anything really vital and conclusive on this highly debatable subject, concerning which an eminent authority is going to speak to you later? I am inclined to think that I may even receive your thanks if I do not venture on this broad field at this time.

The experiences of the recent years during which I have had considerable to do with this Society, and especially for this past year, as I have met with various committees, visited many of the county societies and tried to understand the problems and questions that have come up for consideration, have caused me to ponder about this Society, its organization, its membership, its history, its past policies, its present trend and its future course.

Historically it is an old institution, dating back almost to the year of the signing of the Constitution of this country, and thus it possesses the strength, and perhaps the weakness, of such a tradition.

In size it is small, as such societies run, with less than five hundred members on its roster. We live in a rather sparsely settled and agricultural region, having but few cities, none of them of any great size. I presume that the average doctor works in a place where there are no more than two other doctors close at hand, and it is only occasionally that he sees other medical men than those in his own town. The time and opportunity for such practitioners to meet other doctors, with whom to discuss medical matters, give and receive ideas with respect to their profession, are not very common. Most of them have to be decidedly sufficient unto themselves, and they must carry on

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†Lord, Frederic P.—Professor of Anatomy, Dartmouth College Medical School 1911-. For record and address of author see "This Week's Issue," page 991.

largely on their own momentum, so to speak. Vacations are not frequent or long and are often nonexistent; it is hard to get away from their patients, and for one reason or another they rarely close the office door for more than an occasional day off. Such a life leads to a very real sense of responsibility to their work and their patients, and it also tends to make the doctors somewhat individualistic, relying upon their own efforts, their own judgments and their own ideas and information.

With this background this Society's membership is naturally a more or less loosely bound organization, meeting as a whole but once a year, and in its county units, often not more than twice. Unlike a Rotary Club, for example, which meets every week, it is not reasonable to expect that our smaller groups or the whole Society would be as unified or as active in its functions or policies as the former. And men so occupied with their exacting and arduous daily life, seldom have an opportunity to work shoulder to shoulder with men of their own special interests and training and do not naturally make these rare gatherings a vital factor in their existence. Time is not offered for the exchange of ideas, the formulation of policies, opportunity is not available for ironing out individual differences or making reasonable compromises toward some special end, all of which are necessary for any creative and coöperative results.

The organization of this body, due to its size, and to the few opportunities for assembling, has necessarily been so devised that the greater part of its legislative and deliberative functions is carried out by a group of about thirty men, elected representatives from each local group. Such functions cannot be carried out by a sort of town meeting among several hundred members gathering once a year. The executive work is done by a few special officers and a considerable number of committees. Information of what is done by the House of Delegates and by the special officers and committees is given out by an annual report, issued a year after the meeting has taken place, by monthly publication in a medical journal and at times may be given by special bulletins from the Secretary.

The long history, the character of our membership, and the type of organization of this Society, it seems to me, tend to make it difficult for timely, effective and speedy action where such action may be needed. Also it is not reasonable to expect that such a body would be highly efficient in bringing about a common attitude or its expression on any particular subject.

What the situation has been in the past in this regard is a fair question to ask. Our one gathering each year, such as the present occasion, brings us together to hear a splendid medical program, which we all enjoy, and from which we profit professionally; there is no doubt of that.

Our legislative body meets, adjusts its constitution and by-laws, elects its officers and committees, discusses routine affairs, entertains propositions from its committees, passes resolutions, deplores this or that tendency, recommends certain procedures, some of them of decided value and importance and adjourns for a year, necessarily leaving to its committees the function of carrying out its wishes. So far as the rest of the Society is concerned this usually seems to be the end of it. Some committees meet and work conscientiously, others less so, but all are handicapped by the fact that there seems little general knowledge on the part of the members of the Society, and perhaps too little interest, in what they are trying to do. Little help from among the members seems to be available, considerable opposition seems evident in some cases, and the next year seems to begin about where things were the year before.

I question whether as a group we have not been several laps behind the rest of our State in general; whether we have not preferred to continue things as they have been, without much thought as to what are the demands and wishes of the public, their reasons therefor; whether we have not preferred to let others go ahead, against our opposition or indifference, rather than to study the matters in question, enter into the public discussion, and attempt to use our influence along the lines in which our special training and experience would naturally make us leaders.

If various projects, that have to do with things medical, have been carried on by others than ourselves and in ways which we think are unwise or improperly handled, have we been willing to accept the existing fact that these procedures are in being, and to do our part as citizens as well as specialists in bettering them? Have we been feeling that we belong to a profession which requires perhaps the most laborious and extended period of preparation of any, which carries a tremendous individual responsibility, which is more generous of its time and skill than perhaps any other, and that our position for these reasons is sacrosanct? Have we been feeling that our practices and traditions are to be left as they are; need no adaptation to a changing world, that others should accept our attitude without question, and, if they do not, we should ignore the fact and go on as ever? I ask these questions as being healthy for ourselves, for it is often well that we make examination along the lines where we feel the most secure. Sometimes it comes out under such a process that we can readjust our perspective a little and see even our familiar and accepted path under a different light.

One can easily say that a doctor who is not today carrying on medical practice is not a

suitable person to carry on such queries, let alone to pass judgment on such matters. This is a natural comment to which I can only answer that my recollections of my struggles for several years in the practice of medicine are by no means forgotten; that my regular work is in teaching young men who are to make their living as practicing physicians, and that my deepest interest is and always has been in my profession ever since the days when I first studied medicine. My experiences during this past year with many of the problems that have come to the practitioner have brought close to me their significance to other doctors who are making their living by active practice.

If I am painting a somber and discouraging picture of the New Hampshire Medical Society, it is not with the idea of trying to point out any personal feeling that matters are necessarily hopeless or too discouraging, but rather that I feel there are signs of a very definite improvement already along some lines, and there are possibilities for much further progress. It is for this reason that I have taken the liberty to speak so frankly at the outset.

I want to say at this time that, as I have seen our organization in recent years in a sort of close-up view, I feel certain that we do not lack men, able, informed, interested and willing to work along with the rest toward an improvement of conditions, where such seem to be needed. The heterogeneity and individualism of our Society is not by any means a factor that is altogether harmful or that cannot be overcome where it is a hindrance. What then can be done and along what lines can we make further and more definite progress?

Let me call your attention to certain far-reaching tendencies that have appeared in the last few years in our Society. When this State launched its Relief Program there were many new problems and many new procedures were established, some of which were a cause for great dissatisfaction on the part of our profession. We might have allowed things to remain in that situation, individually, harboring feelings of injustice, or we might have passed a resolution, letting it go at that. Instead there was instituted a committee, headed by our able president of last year, which worked hard and long over the new regulations, doing its best to point out the difficulties, to correct them as much as possible, attempting to inform the medical men of the State as to the meaning of the more vague provisions, and to explain as far as possible where explanation could be made. This committee not only acted as interpreter to our profession, but it went to the State authorities, who had the matter in charge, doing its best to alter and improve in so far as they were able to do so.

At the same time there was introduced another new factor, namely a Special County Committee in each County Society, whose function it was to handle this problem in its details in its own province; to keep in touch with the State Committee of their Society, and at times to clean house in its own County, where evidence was forthcoming that any individual member was not following properly the regulations which the rest of us were trying to carry through.

Many mistakes were made in the administration of this new law, no one will deny that many injustices and inequalities were present, and a deal of unhappy feeling was stirred up. I am not attempting to pass judgment in any way upon Bill 417, or upon its administration in detail, even if I felt more competent to do so. That particular law is a thing of the past. What I do want to call attention to is the fact that in our relation to this law we have set up what is practically a new procedure.

During the past year this device has again been resorted to by our Society in two other similar instances, namely in regard to Child Health and to Maternity and Infancy. Work of a medical nature relating to pre-school and school children, and also to maternal and infant care, is being done by agencies of this State other than our own profession; agencies which we rather presumptuously call lay groups. In both cases there was formed a special committee of this Society whose function was to work with these lay officials. I might add that in one case at least such a committee from our organization was asked for by the lay body which was to carry out the work, in order that they might have the benefit of the advice and help of the medical profession. It is a good guess that such a request, the desire for such coöperation, would never have occurred had it not been for the work of the Committee on State Medical Relief of the previous year.

In the hurried and state-wide work on Child Health, County Society Committees were also established in each of the ten Counties, as in the case of the Welfare work, and for the same reason. The County machinery was new and not always simple to use. While in some Counties it was not availed of, in other Counties these committees were active and made real accomplishments. Again I want to state that I am not attempting here to judge the details of these procedures, but I feel that the plan helped, that there was a very real coöperation, and that in the work of the Department of Education, with the aid of our Society, the task was simplified and friction was lessened by means of our new and somewhat creaky machinery.

It may well be that many or all of these State lay projects were not altogether what they might have been; the work of the various committees was not entirely satisfactory to the members themselves, or to many of the members of this

Society, the difficulties were certainly many. Nevertheless, this development on the part of the New Hampshire Medical Society of coördinating committees of this nature, to work with organizations outside our own is packed with possibilities of the greatest value to the future, both for our organization, and, I dare hope, for the public in general.

There is being brought to the attention of our members at this annual meeting a tentative plan of schedules and regulations, which it is hoped will help to carry out the administration of State Relief in the future. This plan has been worked out by our representatives in conference with the State Relief Commission and representatives of the County Commissioners. Again I do not wish to discuss here the provisions of that agreement—you will hear of them at another time—but I wish to point out that it is the result of a deliberate and careful attempt on the part of our own committee to present the case for the medical profession to the officials having this relief in charge, and an attempt on their part to meet our suggestions and advice as presented to them by active practitioners who know the history of the past two years in this matter and who are acquainted with the opinions and feelings of our profession throughout the State. This sort of coöperation, with the desire on both sides to come to a fair agreement, is inherent in this set-up, and I think we are entitled to feel that it has in it germs of real benefit to all concerned.

Although it may not be entirely satisfactory to any one—what arrangements of this sort ever can be so?—it promises well and carries with it a chance for further alterations where they may be needed. It seems to be the logical and only proper way by which such a measure can be carried along to its fitting conclusion. Where mistakes are made, where injustice is done, there exists through the machinery of the County and State Committees on Relief of our Society an opportunity to call attention to them and to have them considered on their merits, with hopes for remedy.

At this point I want to speak of what I believe is a necessary further step which we must always be ready to take, if we are to do our own part in making such a scheme successful. I think I feel as strongly as does any one here that the members of our profession as a rule are willing to do their part, as they always have in this less remunerative portion of their work, that they are intending to act fairly and to work with the machinery that is set up by the State. If there are any among us who, through misinformation or through deliberate intent, do not carry on their work fairly or who may be tempted to utilize the existing system for selfish ends, I think our organization ought to be willing to correct the one who is misinformed and to censure or penalize the one who is remiss. If we are not willing to do this, we are not doing our part in this group with which the State is working. It means that our County and State Committees may need on occasion to use frank and direct means. I do not surmise that such situations will often arise, but when they do we should be ready to do our part.

The benefits of this procedure where the two parties concerned, the State or lay group and this profession, have gone over the ground together is shown in the recognition of certain factors concerning which there has been much said during the past two years. As regards Welfare for example, the patient, if it is an acute case, is allowed to have the privilege of choosing his own physician; he may receive the first visit without having to wait for special permission from a third party, and as an acute case he may continue with his own doctor at the hospital. The physician does not have to ring up a local official when making his first call on a relief case. Reasonable fees for his work are proposed for the doctor, who in many cases of this nature would formerly have received nothing, and opportunity is offered for adjustment of fees in special cases where question arises.

These provisions represent a reasonable set-up in the care of relief cases by the doctors of this State, and they provide means for further change if, and when, such changes seem desirable. Once more, I am not trying to evaluate in detail the scheme here referred to, but I am calling attention to the fact that this Society has now in existence and actually at work machinery which has been successful to a degree, at least, and which has not as a rule been previously much used or to such good effect. This machinery is capable of further usefulness, both to institute and modify existing arrangements, and similar machinery can be set up for other and new purposes when this is needed.

How does all this bear on the future? We are all well aware that other innovations are being discussed which may affect profoundly the practice of medicine. During the past year the officers of this Society have given much consideration to these problems and have attempted to learn what is the opinion of its members. To that end there have been many meetings of the Committee on Public Relations, Public Policy and Legislation; other members, representing the Society more widely, have been called for a special meeting, discussions have been held among the County Societies, and finally it was thought that these matters were sufficiently important and imminent to call an extra session of the House of Delegates to consider these vital questions.

The officers have attempted, by special bulletins to the members, to call their attention to these problems and some of the evidence bear-

ing upon them, to record the official decisions of the House of Delegates, to interpret to the public our opinions in so far as we can interpret them, and to see that at least the public is aware of the fact that we are interested in such things and are carefully studying them. The officers realize that they are your representatives, and that, even if they should wish to move faster, they cannot rightly make any headway unless the Society as a whole moves with them. The officers are not ashamed of their intent and they have found it difficult going. The rightful interpretation of five hundred minds on burning questions is no simple thing, and if your officers have made mistakes we trust you will tell them.

We may not expect that the various new proposals for Socialized Medicine, Sickness Insurance, etc., will become law, or we may feel that they will surely be in existence within a relatively short time. We may like or dislike them. We may do our best to prevent their coming into being or we may wish to expedite the process. It may be, as some have said, that it will make no difference what we think or do as to the final outcome of such measures. I am not inclined to think that this statement is entirely true.

In so far as we have a collective opinion in regard to the proposed measure of Socialized Medicine let us state these opinions, and do so where it will have the greatest effect. It is not unnatural that we, as a profession, should feel that in such matters we have not only a right to voice our beliefs, but that we have a definite obligation to do so, as being a group who are especially and peculiarly familiar with many of the problems that arise in regard to Sickness Insurance, for example.

What further can we do? I think that what we have already done, the machinery that we have already set up and used, is the clear and self-respecting answer to this question. After the preliminary stages of discussion are over and when this Country or State decides to embark upon any form of Sickness Insurance, then is the time for us to give our advice and co-operation through our constituted representatives.

These representatives will work with the State authorities; we certainly will make an effort to that end—attempting to use our best wisdom in helping to direct the manner in which the laws shall be carried out. Where unfairness and injury are present we can by this means have a real chance to remedy such conditions; to speak for the best interests of those who are most concerned, and to carry on our fundamental work, which is to do all we can for the better health of our patients, who are the people of this State, and who I do not hesitate to say need and, I hope, desire our services.

I hope that this partial attempt to analyze our Society, its history, its membership and its organization, will not be regarded as too unkind or too pessimistic. Such a backward glance seemed to me to be a necessary step in order to bring out our difficulties, and to show the means we have been utilizing to overcome so far as possible these difficulties. It points the way also to what can further be done along these lines toward keeping all of our members better informed; in closer touch with each other and with their elected officials, toward the erection and maintenance of a proper organization which can cause us to be heard, and toward the use of representatives who will keep us in close and friendly touch with the other agencies with whom we may desire to work.

I do not mean that such a device should be used simply to line our own pocketbooks, although I feel it is only fair and right that our interests should be vigorously presented and worked for, but if we physicians wish that our profession be advanced, that public health be best looked after, that we have an opportunity to make ourselves felt through our own organization, it is well that we examine into our own official body as carefully as we do into that of a patient, that we look into all the facts that bear on the case, make our diagnosis and be prepared to institute methods of treatment which will do the most good.

RECENT DEATHS

SNOW—Samuel Dryden Snow, M.D., aged sixty-five, medical referee of Carroll County, was found dead at his home in North Conway on September 20, 1935. He had lived alone since the death of his wife, Louise (Berry) Snow, last December.

Dr. Snow had been a resident of North Conway for the past thirty years, going there originally as resident physician at the tuberculosis sanatorium. He graduated from the Dartmouth Medical School in 1897.

One son, Dryden Snow, who is educational adviser at the Glen CCC camp, survives him.

TOWLE—George H. Towle, M.D., was born in Deerfield, N. H., August 7, 1872, a son of Dr. George H. and Panthia (Tucker) Towle. He received his early education in the Deerfield public schools and prepared for Dartmouth at Coe's Northwood Academy and Tilton School. He studied medicine at Dartmouth and later pursued his medical studies at both Bowdoin Medical School and the University of Vermont. He received his M.D. from the latter institution in 1900.

Dr. Towle had practiced in Newmarket for thirty-three years at the time of his death, October 29, 1935, and had been one of its prominent citizens. He was deeply interested in civic affairs and was a member of the Board of Selectmen. He also served as a member of the School Board, which position he was holding at the time of his death.

Survivors are the widow, a brother in Manchester and a sister in Deerfield.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21461

PRESENTATION OF CASE

The patient, a white American girl about twenty years of age, complained of fever and abdominal discomfort.

She became ill while attending a boarding school and was admitted to the school infirmary with slight fever and malaise. She improved after a few days and was permitted to resume her studies in preparation for graduation. Several days later, however, she again became ill with mild diarrhea, vague upper abdominal discomfort, occasional emesis and complete anorexia. Shortly afterward she developed weakness in both hands and feet which progressed rapidly to a point where she was unable to feed herself. She was sent home, where her temperature ranged daily between 100° and 103° and the pulse varied erratically from 90 to 130. Her gastrointestinal symptoms gradually increased in severity.

At the end of two weeks, physical examination showed a thin, pale, apprehensive girl in no apparent discomfort. There was slight puffiness of her upper eyelids. The ears were normal. The throat exhibited nothing of significance. There were no palpable nodes in the neck. The breasts were normal. Other than a few transient atelectatic râles the lungs were negative. The heart was not enlarged. A soft systolic murmur was heard along the left sternal margin. The blood pressure was 90/64. Examination of the abdomen was negative and no pelvic examination was done. There was bilateral wrist and foot drop with atrophy of the extensor muscles of these parts. The reflexes were normal. The specific reflexes elicited were not recorded.

Examination of the blood showed a red cell count of 3,500,000 to 4,000,000. The white cell count was 14,000 to 23,000. A blood smear showed moderate achromia and an apparent increase in platelets. There were 45 to 60 per cent eosinophils. A stool examination was negative for pus, blood and parasites. Blood cultures showed no growth. The spinal fluid contained two cells per cubic millimeter. There was no increase in the protein content. The globulin and Wassermann tests were negative. Sections of gastrocnemius muscle were negative for trichinosis.

After six weeks the patient's fever fell by lysis but the pulse continued to range widely between 90 and 130. When she had been afebrile for two weeks she was permitted to sit upon the edge of her bed. Gentle massage was instituted for the wrist and foot drop and she was carried out-of-doors once daily. She began to gain weight and obviously became stronger. Her appetite improved although she vomited once and had a short spell of mild diarrhea. One morning she was suddenly seized with a series of convulsive attacks and died before medical attention could be obtained.

DIFFERENTIAL DIAGNOSIS

DR. ALFRED KRANES: This history suggests several possibilities, none of them too convincingly. The first thing that comes to mind, because of the symptoms of generalized infection and bilateral weakness of the hands and feet, is poliomyelitis, of course. There are, however, a number of things here that seem to be somewhat against that. In the first place, it says that she was preparing for graduation. I take it that that was some time in the spring of the year which would make it unusual for poliomyelitis, although it does occur. Secondly, she does not have any headache or pains in the extremities which we associate with poliomyelitis and which most cases do have, and then again the apparent severity of the gastrointestinal symptoms after the paralysis had developed is against it.

Another possibility is poisoning of some kind. Arsenic for instance produces a rather severe gastroenteritis and also peripheral neuritis, but we have no history for that. The onset is rather gradual and not sudden as one would expect with a case of arsenic poisoning. The peripheral neuritis of arsenic poisoning is just as much a sensory neuritis as a motor neuritis, so I think that is probably unlikely.

Can this be a paralysis or peripheral neuritis secondary to deficiency or avitaminosis which we occasionally see in cases of ulcerative colitis? I think that is exceedingly improbable because the peripheral weakness was too marked and there was not enough in the way of gastrointestinal symptoms; not enough diarrhea to account for this degree of weakness. There is nothing else that I can suggest from that history.

It would be a little more helpful if we were told something about the sensory examination. Inasmuch as that is not mentioned here and since she has no sensory symptoms I shall assume that there was no sensory impairment whatsoever. Aside from the bilateral foot and wrist drop the other important feature in that

history is the hypotension which is somewhat unusual for a girl of that age. Poliomyelitis does occasionally produce a hypotension when it involves the vasomotor center, but we have no evidence in this case of any medullary involvement. I think that is probably not the case. The hypotension of course suggests the possibility of Addison's disease. She does have gastrointestinal symptoms, weakness and hypotension but that is all there is in favor of it. There is no pigmentation. The rather rapid onset with fever and the weakness described here is not the usual type of weakness one would expect in a case of Addison's disease; so I think that is also very unlikely.

Nothing is said about the hemoglobin. Of course, the startling thing in the laboratory examination is the very striking eosinophilia. The lumbar puncture, done apparently two weeks after the onset of her illness, does not really rule out poliomyelitis. The spinal fluid might have been abnormal to start with and returned to normal but the other things I mentioned seem to make it improbable. The eosinophilia is very interesting and there are really very few diseases with which I am acquainted which will produce that striking degree of eosinophilia. The most common cause is trichinosis. I think if we knew nothing else except that the patient had 45 to 60 per cent eosinophils and made a diagnosis of trichinosis we would be right in a vast majority of cases. The other, though far more unusual, cause is periarteritis nodosa and there are a number of things in this picture that such a diagnosis might explain.

This is a rather unusual story, a distinctly unusual mode of death for a girl of twenty. I do not know that I can offer any explanation for this picture but I think that in our differential diagnosis we have to keep in mind that this patient was suffering from a disease which affected her gastrointestinal tract, her neuromuscular system, possibly her cardiovascular system, and which in addition produced a fever, leukocytosis and a very high eosinophilia. As far as I know there are only two diseases which are at all consistent with that clinical picture and they are trichinosis and periarteritis nodosa; one a rather common disease of known etiology which is not usually fatal and which produces this variegated picture by the dissemination of the larvae throughout the body by the blood stream, the other a rare disease of unknown etiology which produces very bizarre clinical pictures and is practically always fatal. I think I will have to confine myself to the discussion of these two possibilities. I do not know anything else that would give this picture.

Can it be periarteritis nodosa? She had gastrointestinal symptoms and central nerve involvement and this high eosinophilia. This certainly is not the usual picture of periarteritis nodosa, if one may speak of "a usual picture" in such a rare disease. They usually have a longer course and rarely die within eight weeks as this patient did, usually from four to eight months up to a year and sometimes even longer. So that that seems to be against it. She does not have what most of the patients with periarteritis nodosa have, that is severe pain in the extremities, muscular cramps. She does have what appears to be a peripheral neuritis although we are not quite sure of that. The peripheral neuritis of periarteritis nodosa is mainly a sensory affair and rarely leads to wrist drop and motor signs. The abdominal pain of periarteritis is usually different from this, usually severe abdominal cramps and not mild discomfort and diarrhea. She has none of the urinary findings and none of the cardiovascular findings. They frequently have angina which this patient does not have. Can periarteritis produce this sudden type of death? I think it could with involvement of the cerebral blood vessels. The patient with this disease occasionally dies rather suddenly with profuse subarachnoid hemorrhage; but even with severe subarachnoid hemorrhage one would not expect the patient to die quite so rapidly. On the whole I think that diagnosis is unlikely.

Can trichinosis explain this picture? I do not see anything in there that is inconsistent with that diagnosis. This patient has a number of complaints that people with trichinosis frequently have, abdominal discomfort and diarrhea, fever, malaise and the very high eosinophilia; also puffiness of the eyelids. Is there anything there against the diagnosis of trichinosis? The one thing we usually expect our patients with trichinosis to have is rather severe pain in the extremities, just as in periarteritis, but I have seen a number of them that had no pain whatever. I can recall several who have had no pain but who were later proved to have trichinosis by biopsy and skin test. I do not think that rules it out. As a matter of fact trichinosis is picked up so very commonly when hunted for routinely at postmortem examination that the series ranges all the way from seventeen to thirty per cent in routine autopsy reports. These patients rarely present a history of trichinae infection. How about the negative biopsy? I do not think that rules it out either because we have had patients on whom the diagnosis was eventually established who have had negative biopsies. I recall one we had on the West Medical service who had four biopsies before the diagnosis was established, so that a patient can have trichinosis and a negative muscle biopsy. The unusual bilateral foot and wrist drop we do not associate with trichinosis and yet there have been patients with just that symptom. One patient at the City Hospital was thought to have poliomyelitis for a while and later turned out to have trichinosis. She had bilateral wrist and foot

drop and as one reads the stories of the German epidemics this very severe muscular weakness is not uncommon in severe cases. Some of them become so severe that the patient is unable to sit up in bed not because of pain but because of very profound muscular weakness. So that I think that can fit in with trichinosis. Just what the mechanism of that weakness or paralysis is, is not very clear. Apparently it was not a real peripheral neuritis; that is the nerves themselves are not involved. It seems to be entirely a muscular affair.

If this patient did have trichinosis, how do we account for that rather sudden death? Trichinosis ordinarily does not do that. She died in a series of convulsions which leads us to think there must be some involvement of the central nervous system. Up to that time there was no indication of any involvement at all—negative spinal fluid, no reflex changes and probably no sensory changes, although I cannot be sure of that. We know that the trichinae are occasionally found in the central nervous system. They have been recovered from the cerebrospinal fluid and the brain substance itself and cases have been described where patients died of diffuse encephalitis; but there is no evidence of such a process here. Patients who die with encephalitis in the course of trichinosis usually present the picture of coma, delirium and ocular palsy, none of which this patient had. Trichinosis also produces thrombosis of the peripheral blood vessels and one wonders if it cannot do the same thing in the brain with the cerebral blood vessels and there again I must mention the fact that cases of trichinosis have been described during the course of which the patients have had hemiplegia the nature of which has not adequately been explained. But this is not the picture of cerebral thrombosis and I do not think anything will be found in the brain to account for this type of death. The usual mode of exitus in patients with trichinosis is diffuse myocarditis and we do have some evidence here that there is myocardial involvement. There is that single isolated low blood pressure. It would be extremely interesting to have more blood pressures during the convalescence of this patient. The fact that this patient had a pulse of 90 to 130 while afebrile makes one wonder about the possibility of myocardial involvement in this case. I think it also would be of interest if we had an electrocardiogram some time during the course of the disease because patients with trichinosis frequently show striking cardiographic changes which later return to normal with convalescence. I think this death could have been caused by a sudden acute myocardial failure. If the patient had trichinosis I believe it would be a logical type of death. Trichinae are frequently found in the myocardium if properly digested though not usually in microscopic sections but the myocardial changes are almost always found. That is about the best explanation I have to offer.

Dr. Tracy B. Mallory: We have given Dr. Kranes the compliment of a very difficult case and I think his discussion is so complete there is nothing to add.

Dr. James H. Means: May I add one thing in the list of differential diagnoses? We have seen at least one very bizarre leukemia with extraordinary eosinophilia with cells that were abnormal. I think that is unlikely but I merely mention it as being worthy of the list of diagnoses to be considered.

Dr. Alfred Kranes' Diagnoses

(1) Trichinosis or
(2) Periarteritis nodosa.

Pathologic Diagnosis

Periarteritis nodosa of the coronary arteries.

Pathologic Discussion

Dr. Mallory: I am sorry Dr. Roy Wheeler is not here. This was his case and he himself did the autopsy outside the hospital and sent in some of the organs to me. The heart was normal in size, but as you looked at the surface you could see along the coronary arteries small beads one and two millimeters in diameter. These were separated by intervals of four or five millimeters of normal vessel and then another bead. In cutting through the arteries at the points of beading it was obvious that in some instances they were thrombosed, in other instances not. Microscopically the picture is absolutely typical of so-called periarteritis nodosa. The condition is really a panarteritis, the most important feature of which is necrosis of the media with the formation of miliary aneurysms rather than the periarterial inflammation. A few lesions were also found in some slides of the liver. Unfortunately we did not receive specimens from the other organs, and the brain was not examined.

There is one other possibility of diagnosis in a case of this type that came to my attention within the last year from a very interesting case they had at the Faulkner Hospital with a somewhat similar history. The patient had signs of myocardial weakness with rather more pain in the muscles and rather less weakness than this patient, but some of both. He developed grossly palpable nodules along some of the arteries of the scalp. He had, like this patient, an eosinophilia of 50 per cent. I was told about the case and made of course a snap diagnosis of periarteritis nodosa and urged them to do a biopsy. They did one and the nodules from the scalp vessels proved to be perhaps the prettiest rheumatic nodules I have ever seen. The patient improved for some time and then suddenly died. Dr. Hazzard has al-

lowed me to see sections of the case and there are scattered throughout the heart very numerous Aschoff bodies. There are a few Aschoff bodies to be found in the diaphragm and in the kidney, and there are other lesions that very strongly suggest periarteritis nodosa. On the other hand the lesions of small vessels in rheumatic fever are so similar microscopically to some of the pictures in periarteritis nodosa that it would be difficult to distinguish between the two. I have been unable to make up my mind whether it is rheumatic fever plus periarteritis nodosa or whether an occasional case of atypical rheumatic fever may give that picture.

CASE 21462

Presentation of Case

First Admission. A forty-six year old Finnish laborer was admitted complaining of vomiting and lack of appetite.

He had been well until twelve weeks prior to entry when he suffered from an attack of "grippe" which confined him to bed for two days. The attack consisted only of malaise but at its termination he developed an abscess in the right groin which was incised by his physician. It continued to drain purulent material for three weeks after which it healed spontaneously. He developed anorexia which was associated with nausea, occasional emesis and a sensation of light-headedness. He returned to bed shortly after his abscess was incised and remained there for nine weeks. Vomiting, which at first occurred irregularly, increased in frequency until he vomited two to three times each day. During the ten weeks before admission he developed a nocturia of two to three times. Two weeks before admission he began to vomit everything ingested. Slight swelling of the ankles appeared. There were occasional attacks of "dizziness". He became progressively weaker and developed slight dyspnea on exertion. In the three months preceding his admission his weight had dropped from 165 to 135 pounds.

He had had incision and drainage of a left inguinal abscess forty years before entry. Seventeen years later he was seen in the Outpatient Department where he presented a story of morning cough with blood-tinged sputum and pain in the chest of two years' duration. Inconstant râles at the right top and slight dullness of the left were found on examination. These findings were not confirmed subsequently and the cough gradually subsided. Because of multiple caries and apical abscesses his teeth were extracted three years before entry.

Physical examination showed a well-developed but poorly nourished man with evidence of recent weight loss. His skin was dry and scaling and there was fairly marked pallor of the mucous membrane. The heart was negative except for slight accentuation of the aortic second sound. The blood pressure was 110/90. There was a sharp scoliosis of the third to fifth dorsal vertebrae with a convexity to the right. The lungs were clear. The liver edge was irregular and was found to be 3 centimeters beneath the costal margin in the midclavicular line. An ill-defined globular mass which moved slightly with respiration was felt in the epigastrium just above the umbilicus. (Neither the abdominal mass nor the liver edge was demonstrable at subsequent examinations.) There were recent scars in the right inguinal region and old healed scars in the left. An eczematoid eruption was observed on the right lower leg.

The temperature was 98.6°, the pulse 84. The respirations were 20.

Examination of the urine showed a specific gravity of 1.008 and a large trace of albumin. The sediment contained three to five white blood cells, a rare red blood cell and an occasional hyaline and granular cast. A urine concentration test exhibited fixation of the specific gravity at 1.008 to 1.009. The blood showed a red cell count of 4,900,000, with a hemoglobin of 70 per cent. The white cell count was 9,500, 71 per cent polymorphonuclears. The stools were negative. A Hinton test was negative. The nonprotein nitrogen of the blood was 80 milligrams, the serum protein 4.1 grams per cent. The chlorides were equivalent to 110 cubic centimeters N/10 chloride per 100 cubic centimeters. An intravenous phenolsulphonephthalein test showed excretion of 10 per cent of the dye in two hours. An intracutaneous injection of old tuberculin in a dilution of 1:40,000 was negative. The icterus index was 2. A liver function test showed 0 to 5 per cent retention. The van den Bergh was normal.

An intravenous pyelogram was interpreted as demonstrating non-concentrating kidneys of normal size. An x-ray of the gastrointestinal tract exhibited no abnormality of the esophagus, stomach or duodenum. A barium enema showed ballooning of the rectum, spasm of the sigmoid and unusually poor emptying. No lesions could be demonstrated.

The patient continued to vomit solid food at intervals but his appetite improved. On the fifth day another phenolsulphonephthalein test showed no excretion of the dye in two hours. The nonprotein nitrogen of the blood was 68 milligrams. The red blood cell count was 3,600,000, with a hemoglobin of 80 per cent. He was given glucose intravenously and tincture of belladonna by mouth and the vomiting eventually ceased on the eighth day. The temperature, pulse and respirations remained normal throughout the hospital stay. His anorexia

and weakness subsided and he was discharged home at the end of the second week.

Second Admission, two and a half months later.

Shortly after his return to his home he began to vomit again with progressively increasing frequency. He became quite weak and developed an intolerable generalized itching of the skin. He was somewhat drowsy at times. Two weeks before entry a tender swelling had appeared on his right cheek at the corner of the mouth. This had grown larger, ruptured externally and exuded purulent material.

Physical examination showed a sick looking, pale, drowsy, air hungry man who scratched himself continually. The skin was dry and scaly and contained a few brownish blotches. There were scattered deposits of fine white granular material on the skin surface. Retinal edema was observed in the left fundus. Examination of the right side was not recorded. An abscess draining white creamy pus was present at the right corner of the mouth. There was an indurated tender swelling at the left corner of the mouth also. The lungs were clear. The heart was not enlarged and the sounds were distant. A basal systolic murmur was heard. The pulse was feeble. There was no edema. The blood pressure was 125/90.

The temperature was 98.8°, the pulse 95. The respirations were 20.

Examination of the blood showed a red cell count of 2,400,000, with a hemoglobin of 50 per cent. The white cell count was 20,000. There were 76 per cent polymorphonuclears, 11 per cent lymphocytes and 13 per cent eosinophils. The blood chlorides were equivalent to 115 cubic centimeters N/10 chloride per 100 cubic centimeters. The serum protein was 4.6 grams per cent, the nonprotein nitrogen of the blood 150 milligrams per cent, and the carbon dioxide combining power was 20 volumes per cent.

The patient was given 2,000 cubic centimeters of 10 per cent glucose solution intravenously. He failed rapidly, his temperature dropping to 97°, and died thirty-six hours after admission.

Differential Diagnosis

Dr. Fuller Albright: The whole problem in this case will be to find out why he has renal insufficiency, what type, not whether he has that condition. The vomiting and lack of appetite will turn out, as we read more of the history, to be obviously due to chronic uremia.

Since his illness started with infection you immediately think of glomerulonephritis as opposed to other types of renal disease.

With a draining abscess in the groin one cannot help thinking of lymphogranuloma inguinale, which recently seems to be turning up on all sides in this hospital. It may be due to an old tuberculous gland or anything else.

With all these recurrent abscesses we are now thinking a little bit of amyloid.

Low blood pressure is against chronic glomerulonephritis.

An indefinite mass in the epigastrium usually means nothing.

"The sediment contained three to five white blood cells, a rare red blood cell and an occasional hyaline and granular cast." This pretty definitely puts it in a chronic nephritis group as opposed to various urological conditions.

We are surprised to see that the red cell count was normal.

"The nonprotein nitrogen of the blood was 80 milligrams, the serum protein 4.1 grams per cent." A kidney that has difficulty in excreting nonprotein nitrogen, and does not hold back protein. The chlorides are elevated. You would expect this value to be slightly high with the serum protein so low.

He has a very low renal function.

I do not think there is any question but that our trouble is with the kidney. I do not think he has uremia due to vomiting and dehydration alone. I think from the kidney findings that the pathology must be in the kidney. I think that all these other studies are aside from the point.

"The skin was dry and scaly and contained a few brownish blotches." Some superficial infection I imagine.

"Retinal edema was observed in the left fundus." The fundi are certainly surprisingly lacking in change.

There is no evidence of hypertension as yet. He now has very marked anemia. He undoubtedly had a lot of infection. It is not unlikely that a terminal septicemia or something set in.

It is a little hard to explain why the blood chlorides should be up to 115. He had marked terminal acidosis as so many of these patients have. I am surprised to see the chlorides that high.

I should think the most likely thing that Dr. Mallory will tell us is that the kidney is of the same type primarily, as that in Dr. Baker's case, a kidney about normal in size, the so-called pale white kidney,—a chronic glomerulonephritis that went through a nephrotic stage and ended in typical uremia with these infections to contribute. I should not be too surprised if he told us he had amyloid disease. Consistent with this were the very low protein, the terminal rise in blood nonprotein nitrogen, and absence of eyeground changes. He had perhaps an etiology for amyloid. I think we have to consider the other insidious conditions that will destroy kidneys; multiple myeloma certainly is to be considered but with that you would expect a high serum protein rather than low. Hyperparathyroidism has to be considered but I think I would have heard of it if it had been hyperparathyroidism.

X-ray Interpretation

Dr. George W. Holmes: There are one or two things that I might point out. As Dr. Albright suggested, there is nothing of particular importance in the x-ray. You can see the kidney outlines on both sides and although we are not able to state definitely the size of the kidney from x-ray film, you can detect gross changes. The size here appears to be normal. This is the edge of the liver and it does not seem to be enlarged. Again the x-ray evidence of enlargement of the liver is of uncertain value. The question was raised as to the presence or absence of trouble with the spine. We have lateral and anteroposterior views of the spine; they show scoliosis but no evidence of disease. Here is the film of the chest which also shows quite marked scoliosis but no evidence of disease of the lungs. In other words, no primary tuberculous infection of the lungs, nothing to suggest that the other lesions were of tuberculous origin.

Clinical Discussion

Dr. Alfred O. Ludwig: When this man first came into the wards there was a discussion as to what the diagnosis might be because of the anemia and blood pressure. We finally felt that it was chronic glomerulonephritis. He was followed for a time in the Outpatient Department and later we sent him into the house again with the opinion that he was in a terminal stage of chronic glomerulonephritis and some secondary infection. We still were concerned at that time that the blood pressure was not higher. He lacked eyeground changes.

Clinical Diagnoses

Chronic glomerulonephritis.
Uremia.

Dr. Fuller Albright's Diagnoses

Chronic glomerulonephritis.

Anatomic Diagnoses

Chronic amyloid nephrosis, with contracted kidneys.
Bronchopneumonia.
Lymphangiectasis of the small intestine.

Pathologic Discussion

Dr. Tracy B. Mallory: Very obviously there are discrepancies in the clinical picture. The diastolic blood pressure is high, but the systolic is quite low. The serum proteins are certainly very low for a chronic glomerulonephritis, and he did show typical retinitis.

At autopsy we found moderately shrunken kidneys. The pair weighed 150 grams. On the other hand the surface was only slightly granular, not so markedly so as one would expect, the tissue was rather firm and had a slightly waxy consistency. The sections have served only to increase the mystery a bit more. The picture is a peculiar one in that the most striking feature of the kidney is the persistence of glomeruli, which are all hyalinized, and almost complete absence of convoluted tubules. Ordinarily when the glomerulus is sclerosed and hyalinized in chronic nephritis, it eventually disappears, the time of disappearance probably taking two or three years. As it loses its function the corresponding tubules atrophy and disappear, and one is left with a fibrous scar which is usually rather densely infiltrated with lymphocytes. Not only the convoluted tubule but the corresponding Henle's loop and collecting tubule disappear. In this man, however, Henle's loops were well preserved, the collecting tubules seem actually to have proliferated a bit and the convoluted tubules are all gone. With ordinary stains the glomeruli are apparently characteristic of amyloid disease but with the amyloid reaction they give a very feeble and not entirely characteristic stain. There is no amyloid in the follicles of the spleen, the commonest place, and none in the liver. On the other hand the adrenals are full of amyloid. The renal picture suggests that at some time in the past something has specifically knocked out the convoluted tubules. The findings seem to fit the assumption of a contracted kidney based on an old tubular nephritis, a diagnosis that was often made fifteen years ago but which has gone completely out of fashion now.

It should be remembered, moreover, that in amyloid nephrosis the tubules regularly show more or less severe lipoid degeneration, a condition which might well be followed by atrophy. Fahr, as a matter of fact, describes "amyloid Schrümpfniere" in which the shrinkage is due to tubular atrophy, and such a diagnosis is, I think, our best bet on this case.

This man did have some pathology in the gastrointestinal tract in the form of multiple polypoid projections which on section seem to be greatly dilated lymphatics in the small intestine. He had a terminal bronchopneumonia, a normal sized heart. The other organs were essentially negative.

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COLLEGIATE PREMEDICAL EDUCATIONS

THE proposal of the Board of Registration in Medicine that the requirements for premedical education shall be raised from mere high school to include two years of collegiate work takes one back to 1918 when this requirement was generally adopted for approved medical schools. The previous formal step had been taken in 1914 when one year of college became the requirement.

It is interesting to note that although the evaluating bodies whose opinion is generally accepted have not raised their formal requirements since 1918, many medical schools require more than two years of premedical collegiate work for their own students and a very considerable number of candidates for medical school present four years of college credits and the baccalaureate degree. This raising of the informal requirements so high above the formal, is due to the great demand for medical education, far greater than medical schools now in existence can supply.

The specific content of the two collegiate years is often not stated except that it shall include physics, chemistry, and biology. These subjects were formerly taught, in so far as they were taught, in the medical curriculum. At least as far back as 1893, they came to be formally, though not universally, relegated to the premedical schools. By 1922, the conviction had become so widespread, that the physician should have at least an acquaintance with these fundamental sciences, that Massachusetts became aware of the situation, and there were introduced into the list of statutory subjects for examination these three, physics, chemistry and biology. As a high school education became a requirement at the same time, a mere casual familiarity with science was deemed sufficient.

If one reviews the laws of the states, one finds that only eight have failed to make statutory provision for the two years of college. In six of the eight, however, the boards of registration have, under the statute and in the exercise of their discretion, refused to accept graduates of schools which do not have this requirement. Massachusetts stands almost alone in clinging to the low standards which, over forty years ago, nearly half a century, enlightened institutions outgrew.

With the progress of knowledge, the burden of knowledge and training on the medical student and the physician has become heavier, to such an extent that few, if any, are able to become well-qualified physicians without the formal education now generally required. The day has gone by, when anyone without formal training in physics, chemistry and biology can provide for himself an adequate foundation for his work in the medical school.

There is another consideration to which more explicit attention should be given. Why has medicine made such rapid progress in recent years? The reasons may be summarized briefly in the statement that the closer association of the university departments of these premedical sciences with the medical schools and the application of their ideas to medicine have resulted in a remarkable fertilization in both the preclinical sciences and in their application in practice. Indeed the result of the impact of science on the art of medicine in the past twenty-five years has been one of the remarkable phenomena of the twentieth century. It is now impossible to understand modern medicine without insight into the introductory and basic sciences.

Since this formal education in science is necessary and is so regarded by the State, Massachusetts should also require that it be more nearly adequate, that is, of collegiate grade.

CELSUS *DE MEDICINA*

Of the three great medical figures of the Classical Period—Hippocrates, Galen and Celsus—the least is known about the life of the latter. The author of a great medical treatise, which profoundly affected the practice of medicine throughout the ages and even down to our time, may not have been even a practitioner of the art. Quintilian states that he wrote on many other subjects than medicine and from this statement it has been inferred that Celsus was merely a general editor of an encyclopaedia, perhaps not even the author of *De Medicina.* Dr. W. G. Spencer, who has recently translated the first four books of *De Medicina** takes the view, based on a number of passages in his work, that Celsus was "a learned and experienced medical practitioner . . . even allowing for the less sharp distinction drawn between the professional and amateur in his day." The book would "seem to indicate that he himself regularly attended patients, and wrote with the authority of a practising physician."

Whatever views we may hold about the authorship of *De Medicina,* all agree that the book has had great influence as a text of medical practice. A number of manuscripts exist testifying to its popularity before the days of printed books. Before 1500, moreover, it was printed four times, the *editio princeps* at Florence in 1478. Three of these incunables are in the Boston Medical Library in the Bullard Collection. When printing became more common, edition after edition came off the presses, forty-nine before 1841. The best translations, before Spencer's, were those by James Greive, in many issues, and by G. F. Collier. Required reading for applicants for license by the Royal College of Surgeons, many interlineal translations were printed in the eighteenth and nineteenth centuries in England. Scholarly comments on Celsus have been written by Daremberg, Marx and others; much of interest about the man, his book and his time will be found in Allbutt's *Greek Medicine in Rome* and in Payne's article on Roman medicine in the *Cambridge Companion to Latin Studies.* Finally we have the readable translation by the London surgeon, W. G. Spencer, a worthy addition to that section of the medical historian's library of *Celsusiana.*

*Celsus *De Medicina,* with an English Translation by W. G. Spencer. In two volumes. Volume I. Loeb Classical Library. Cambridge, Mass.: Harvard University Press, 1935. XIV, 499 pages.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Derow, Harry A. M.B., M.D. Boston University School of Medicine 1927. Assistant Physician, Beth Israel Hospital. Instructor in Medicine, Harvard University Medical School. Address: 475 Commonwealth Avenue, Boston. Associated with him is

Altschule, Mark D. B.S., M.D. Harvard University Medical School 1933. Assistant in Medicine, Harvard University Medical School. Resident in Medical Research, Beth Israel Hospital. Address: 330 Brookline Avenue, Boston. Their subject is "Malignant Hypertension." Page 951.

Shedden, William M. A.B., M.D. Harvard University Medical School 1920. F.A.C.S. Assistant in Anatomy, Harvard University Medical School. Instructor in Surgery, Tufts College Medical School. Assistant Surgeon to Out-Patients, Massachusetts General Hospital. Chief of Tumor Clinic, Boston Dispensary. Address: 270 Commonwealth Avenue, Boston. Associated with him is

Johnston, Fulton. M.D. Harvard University Medical School, 1927. Member of the Emerson Hospital Staff. Address: 8 Sudbury Road, Concord, Mass. Their subject is "Traumatic Rupture of the Liver." Page 960.

Day, Hilbert F. Ph.B., M.D. Harvard University Medical School 1905. F.A.C.S. Professor of Clinical Surgery, Tufts College Medical School. Surgeon-in-Chief, Boston Dispensary. Senior Surgeon, Cambridge Hospital. Surgeon, Boston Floating Hospital. Consulting Surgeon, Massachusetts State Prison Colony, Norfolk, Mass. His subject is "The Organization of a Varicose Vein Clinic." Page 966. Address: 412 Beacon Street, Boston.

Whitney, Edward T. A.B., A.M., M.D. Harvard University Medical School 1924. Assistant Surgeon, Boston Dispensary. Surgeon, New England Telephone and Telegraph Co. Address: 587 Beacon Street, Boston. Associated with him is

Consales, Peter A. M.D. Tufts College Medical School 1922. Junior Surgeon, Boston Dispensary. Address: 481 Beacon Street, Boston. Their subject is "The Treatment of the Varicose Ulcer." Page 967.

Whitney, Edward T. Record appears above. His subject is "The Treatment of Phlebitis." Page 970.

Levenson, Walter S. A.B., M.D. Harvard University Medical School 1922. F.A.C.S. Assistant in Surgery, Harvard University Medical School. Instructor in Surgery and Anatomy, Tufts College Medical School. Surgeon, Out-Patient Department and Junior Visiting Surgeon, Beth Israel Hospital. Surgeon, Boston Dispensary. Assistant Surgeon, Boston Floating Hospital. His subject is "High Ligation in the Treatment of Varicose Veins." Page

972. Address: 370 Commonwealth Avenue, Boston.

HORLICK, S. SEYMOUR. A.B., M.D. Tufts College Medical School 1928. Junior Assistant Surgeon, Boston Dispensary and Beth Israel Hospital. His subject is "The Multiple Injection Method of Treating Varicose Veins." Page 973. Address: 1860 Commonwealth Avenue, Boston.

BARNEY, J. DELLINGER. A.B., M.D. Harvard University Medical School 1904. F.A.C.S. Chief of Service, Urological Department, Massachusetts General Hospital. Assistant Professor of Genito-Urinary Surgery, Harvard University Medical School. His subject is "Personal Experiences with Tumor of the Bladder." Page 976. Address: 87 Marlboro Street, Boston.

LORD, FREDERIC P. A.B., M.D. Dartmouth College Medical School 1903. Professor of Anatomy, Dartmouth College Medical School 1911-. President, New Hampshire Medical Society 1934-1935. His subject is "President's Address." Page 979. Address: 39 College Street, Hanover, New Hampshire.

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CONTRACEPTION AND STERILITY

One of the striking things that appears in the study of sterility is the increase in its frequency which has occurred during the last decade or two. Statistics are unreliable at best but it seems fair to say that there has been an increase from 10 per cent to 15 per cent, or even 17 per cent. Such a change deserves careful study. Unfortunately the statistical sources of our information are so uncertain as to be practically worthless, but the impression which one gathers from talking with both members of many sterile couples may at least indicate the trends, if not the causes, of this phenomenon.

Of these sterile couples there is hardly a single one nowadays that has not employed some measure designed to prevent conception. Twenty years ago this was not the case though even at that time probably 50 per cent, or two-thirds of the couples, had done so. This immediately raises the question whether the prolonged use of any contraceptive technique, no matter how carefully employed, may not, by denying the natural changes in the woman's pelvic organs which pregnancy initiates, produce a sort of functional sterility. It is my firm belief that it does. It is also my belief that the longer birth control has been practiced, the longer this period of functional sterility will last. This is a common experience with those couples who eventually do conceive without assistance. In most instances it is temporary but in some, where a minor catarrh develops in the cervical canal, where there is congestion from a retroversion, or where other such minor conditions arise, such a functional sterility becomes permanent unless treated. The great increase in numbers of women who, though menstruating regularly, rarely ovulate would seem to postulate hormone changes as possibly resulting from a denial of pregnancy while the sexual stimulation of marriage still persists. In other cases changes in the life of the woman from great activity in business to the relative ease of keeping house for two people, together with dietary indiscretions, predispose toward great changes in nutrition or metabolism, and this often affects fertility. Then, too, there is the effect of time and business cares on male fertility. In many cases of which we have careful records male fertility has decreased to a dangerously low level under the stress and strain of modern business conditions in a relatively short time.

If we can accept these trends as following from prolonged use of birth control early in marriage, it would seem that we as physicians had a duty to broadcast this information. As far as one can judge from numerous conversations with young people who are about to become married there is practically speaking not a single couple, unless forbidden by religion, that does not contemplate postponement of having a family. Quite apart from whatever our attitude may be as to the morality of such a course, it would seem imperative for us to protest against the possible consequences of sterility and ill health which may, and often do, follow.

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

THIRD ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning November 17:

Barnstable

Sunday, November 17, at 4:00 P.M., at the Cape Cod Hospital, Hyannis. Subject: Kidney and Bladder Diseases, A (Medical): Acute Nephritis — Etiology, Diagnosis and Treatment. Nephrosis and Its Treatment. Instructor: L. B. Ellis. J. I. B. Vail, Chairman.

Bristol South (Fall River Section)

Monday, November 18, at 4:00 P.M. at the Stevens Clinic of the Union Hospital, Fall River. Subject: Lung Diseases: (a) Differential Diagnosis and Treatment of Lobar Pneumonia. (b) The Surgical Problems of Empyema. Instructors: Maxwell Finland and H. F. Newton. Eugene A. McCarthy, Sub-Chairman.

Essex North

Friday, November 22, at 4:00 P.M., at the Hotel Bartlett, 95 Main Street, Haverhill. Subject: Kidney and Bladder Diseases, A (Surgical): Hematuria: Its Significance in Surgical Diseases of Kidney and Bladder. Instructor: F. H. Colby. Francis W. Anthony, Chairman.

Essex South

Tuesday, November 19, at 4:00 P.M., in the Nurses' Home of the Salem Hospital, Salem. Subject: Diseases of the Liver: Surgical Problems in Diagnosis of Acute Disease of Gall Bladder and Liver. Instructors: C. M. Jones and L. S. McKittrick. Walter G. Phippen, Chairman.

Hampden

Thursday, November 21, at 4:00 P.M., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 P.M., at the Holyoke City Hospital, Holyoke. Subject: Lung Diseases: (a) Differential Diagnosis and Treatment of Lobar Pneumonia. (b) The Surgical Problems of Empyema. Instructors: R. H. Sweet and D. S. King. George L. Schadt and George D. Henderson, Chairmen.

Hampshire

Wednesday, November 20, at 4:15 P.M., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Pediatrics: Abdominal Disease in Childhood; Medical and Surgical Aspects. Instructors: P. H. Sylvester and T. H. Lanman. Robert B. Brigham, Chairman.

Middlesex South

Tuesday, November 19, at 4:15 P.M., at the Cambridge Hospital, Cambridge. Subject: Cancer of Breast and Uterus; Sarcomas of Bone; Lymphoma and Leukemia. Their Early Diagnosis. Discussion of Life History of Cancer and Grades of Malignancy. Instructors: J. V. Meigs, C. E. Dumas and Dudley Merrill. Edmund H. Robbins, Chairman.

Norfolk South

Monday, November 18, at 8:30 P.M., at the Quincy City Hospital, Quincy. Subject: Pediatrics: The Neonatal State and Its Diseases; Medical and Surgical Aspects. Instructors: J. L. Morse and W. E. Ladd. David L. Belding, Chairman.

Plymouth

Tuesday, November 19, at 4:00 P.M., at the Brockton Hospital, Brockton. Subject: Arthritis: (a) Medical Care of Patient in the Home. (b) Orthopedic Treatment in Hospital and Aids in Home Treatment. Instructors: R. T. Monroe and W. T. Green. W. H. Pulsifer, Chairman.

Worcester North

Friday, November 22, at 4:30 P.M., at the Burbank Hospital, Fitchburg. Subject: Psychiatry: Management of Psychic States in the Care of General Diseases, Especially Chronic Disorders. Subpsychotic States. Instructors: G. C. Caner and Maurice Fremont-Smith. Edward A. Adams, Chairman.

MISCELLANY

MEDICAL HISTORICAL PAGEANT

A public performance of the fifth annual medical historical pageant presented by medical students of Tufts College Medical School, under the direction of Dr. Benjamin Spector, will be given at the Boston Medical Library on Monday evening, November 18, at eight o'clock.

The performance will be under the auspices of the Boston Medical History Club and a demonstration of manuscripts and books having to do with the characters in the play will be made by James F. Ballard, Director of the Boston Medical Library.

These pageants are unique in their method of preparation and presentation. The students write their own scripts after parts have been assigned to them by Dr. Spector. The characters are costumed in the correct dress of the periods which they represent.

The current pageant has to do with the quotation "Story of Early Medicine in Massachusetts", which is divided into three epochs; the Colonial (1620-1700), the Pre-Revolutionary period (1700-1775), and the Post-Revolutionary period (1775-1846).

In the Colonial Period will be found the Indian Medicine Man, Deacon Samuel Fuller of Plymouth, the two Governors Winthrop and the Reverend Thomas Thatcher of Boston.

The Pre-Revolutionary Period is subdivided into Smallpox Inoculation, Midwifery and Obstetrics, and Apprenticeship in Medicine.

The Post-Revolutionary Period is also subdivided into Cowpox Vaccination, the Ether Demonstration, and Oliver Wendell Holmes and Puerperal Fever.

All interested persons and their friends are cordially invited to attend the performance at the Boston Medical Library.

A RESOLUTION ADOPTED BY THE MASSACHUSETTS MEDICO-LEGAL SOCIETY

Whereas, The sale by druggists and dispensing by physicians of strychnine alone or incorporated with other drugs in laxative and tonic tablets with

chocolate or sugar coatings has resulted in many fatalities among children, innocent of their potency and attracted by the candied coatings, and

Whereas, The known therapeutic value of strychnine in no wise justifies such loss of life, suffering, and sorrow,

Be it, by the Massachusetts Medico-Legal Society in regular meeting assembled

Resolved, 1. That it go on record as discountenancing the sale or dispensing of strychnine in any form which covers or conceals its taste or without being clearly labeled as to name, amount, in each dose, and the warning "poison" if dosage is exceeded, and treatment if excess is accidentally taken.

2. That the copies of this resolution be published in The *New England Journal of Medicine* and that the Massachusetts Pharmaceutical Society and the Massachusetts Medical Society be invited to coöperate in preventing the sale or dispensing of this drug in concealed, dangerous form.

October 2, 1935.

ANTERIOR POLIOMYELITIS CASES REPORTED FOR THE WEEK ENDING NOVEMBER 9, 1935

City or Town	No. of Cases
Fall River	2
Brockton	3
Belmont	2
Boston	4
Chelsea	1
Concord	1
Malden	1
Melrose	1
Newton	2
Lawrence	1
Lynn	4
Methuen	2
Middleton	1
Worcester	1
Total	26

Total Cases for Year

January-June	14
July	46
August	481
September	537
October	237
November	34
Total	1349

HEALTH SURVEY*

Memorandum for the Massachusetts Medical Society

Plans have been prepared for a survey of the extent of disabling illness and a study of public health and medical facilities in nineteen States. The survey is to be conducted under the auspices of the United States Public Health Service as a project financed by the Works Progress Administration.

The survey follows closely the well-established policy of the Public Health Service and employs essentially the same methods which have long been used by the Service. It is, in fact, a continuation of activities which have been carried on from time to time over a period of nearly twenty years. Examples of these activities are:

1. Surveys of State, city and county health departments at the request of State or local communities.
2. Epidemiological studies. Frequently these studies have embraced a more detailed inquiry into economic, industrial, racial, and other influences affecting the spread of disease than that contemplated in the present study.
3. Numerous reports based upon physical examinations of school children and industrial workers to determine the extent of disease and physical impairment. These studies were limited to certain diseases and to certain groups of population and, therefore, only partially depict the true health situation.
4. Morbidity studies. These studies were first undertaken fifteen years ago and relate to selected groups of the population or to selected areas. In the first as well as the later studies local physicians coöperated fully with the Service in checking statements concerning the nature of illness reported to field enumerators by the families surveyed.

The foregoing activities are embodied for the most part in the larger and more comprehensive surveys now being made. Methods that have proved successful in the activities listed above have been adapted and refined for application to that part of the present study which has to do with the collecting of data on the prevalence and distribution of the chronic disabling illnesses among representative groups of the general population. Until now such data have been very meagre or wholly lacking.

The program has been prepared and revised following a conference held by the Surgeon General with Drs. Leland and Bauer of the American Medical Association.

The purpose of this memorandum is to acquaint the members of your Society with the objects of the survey and solicit their coöperation in the checking of statements concerning illnesses reported to our enumerators. The coöperation of the medical profession in the areas selected for study is vital to the successful conduct of the study and to the ultimate value of the material collected.

A typical schedule will be sent to the family physician with the request that he check the precise or provisional diagnosis. Physicians will receive

*Submitted by the Public Health Service.

twenty-five cents for each schedule properly filled out.

NOTE: A copy of the schedule may be consulted, in advance of its distribution, at the office of Dr. Begg, 8 Fenway, Boston, Mass. It also appears in the *Journal of the American Medical Association*, October 5, 1935.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR SEPTEMBER, 1935

Disease	Sept., 1935	Sept., 1934	5 Yr. Average*
Anterior Poliomyelitis	540	8	159
Chicken Pox	99	101	89
Diphtheria	19	37	95
Dog Bite	793	756	589
Epidemic Cerebrospinal Meningitis	7	5	6
German Measles	46	19	24
Gonorrhea	553	556	612
Lobar Pneumonia	105	90	87
Measles	51	43	88
Mumps	215	75	99
Scarlet Fever	218	273	305
Syphilis	462	284	321
Tuberculosis, Pulmonary	261	276	295
Tuberculosis, Other Forms	33	38	41
Typhoid Fever	14	22	29
Undulant Fever	3	1	
Whooping Cough	254	506	474

RARE DISEASES

Anterior poliomyelitis — 540 cases (see weekly lists).

Dysentery (amebic) was reported from Fitchburg, 1; Middleboro, 1; total, 2.

Dysentery (bacillary) was reported from Boston, 5; Falmouth, 1; Worcester, 1; total, 7.

Encephalitis lethargica was reported from Lynn, 1; Uxbridge, 1; total, 2.

Epidemic cerebrospinal meningitis was reported from Boston, 1; Cambridge, 1; Salem, 1; Shrewsbury, 1; Stockbridge, 1; Taunton, 1; West Bridgewater, 1; total, 7.

Malaria was reported from Chelsea, 1; Revere, 1; total, 2.

Pellagra was reported from Boston, 2.

Septic sore throat was reported from Boston, 9; Lynn, 1; Mansfield, 1; total, 11.

Tetanus was reported from Great Barrington, 1; Medford, 1; Salem, 1; total, 3.

Trachoma was reported from Boston, 2; Cambridge, 1; total, 3.

Trichinosis was reported from Arlington, 1; Boston, 1; Falmouth, 1; total, 3.

Undulant fever was reported from Auburn, 1; Clinton, 1; Worcester, 1; total, 3.

*Based on the figures for the preceding 5 years.

Infantile paralysis is now on the decline, having had a September peak.

Diphtheria, with the lowest monthly figure ever reported, is running 50 per cent lower than in 1934.

Lobar pneumonia for the sixth month in a row was reported slightly over last year's figure.

German measles had a September morbidity somewhat over the five-year average. It may be that we are in for another year of increased incidence.

Mumps is running higher than in 1934.

Measles, scarlet fever, typhoid fever, pulmonary tuberculosis, tuberculosis other forms, and whooping cough were all reported at a lower level than in 1934.

Chicken pox and epidemic cerebrospinal meningitis were not remarkable.

CORRESPONDENCE

ELECTROSURGICAL CHOLECYSTECTOMY

Editor, *New England Journal of Medicine*,

In the October issue of your valued *Journal* (No. 14) there appeared an article by Dr. Lester R. Whitaker on "Electrosurgical Cholecystectomy" which discusses my operation. Accuracy demands that I correct certain statements in the article referred to, and I hope these comments will be allotted space in your valued columns.

Whitaker used a modification of Pribram's procedure in sixteen cases. His results disclose an operative mortality of 18.45 per cent, 31 per cent of infections, and above 50 per cent of bile leaks, a decidedly worse record than is obtained from removing the gallbladder with the knife which shows about 2 per cent mortality in selected and a global mortality of 9.6 per cent in complicated cases.

In other words, twice as many people succumbed at the hands of Whitaker as in the worst cases operated on by surgeons elsewhere. This statement is based on the exhaustive studies of 12,144 cases of Enderlen and Hotz.

I regret to note that in his discussion of my operation (which, by the way, I have performed to date on 149 consecutive unselected patients without a single fatality), Dr. Whitaker failed to grasp the rationale and principles underlying my procedure which are as follows:

1. An understanding of the differences between fulguration, carbonization and coagulation is a sine qua non.

2. The appreciation of the fact that drains are inimical to electrosurgically treated surfaces. This has been pointed out time and again by a number of observers (Kuntzen and Vogel, and others) and corroborated by my own researches. Even Pribram, whose method Whitaker used, admonishes *not to drain*. I believe Whitaker's disregard for this fundamental rule is in a great measure responsible for his failures.

Electrocoagulated surfaces within the abdomen agglutinate with contiguous serous surfaces within about three hours after operation. If a drain is introduced, agglutination is frustrated and infection ensues. Electrocoagulated surfaces within the abdomen heal by encapsulation and absorption. Superimposition of the falciform ligament, as is practiced in my operation, helps in accomplishing this result[1], [2], [3].

Experience contradicts Whitaker's conclusions on the behavior of electrocoagulated tissues.

3. Whitaker's observations are diametrically opposed to those of Baker on the question of postoperative biliary leakage.

4. Whitaker has apparently not read Schörchers exhaustive studies on the "why" of the absence of shock in electrosurgical maneuvers[4].

5. In discussing my operation Whitaker says, "Thorek depends upon the collapse and coagulation of the vessel walls to prevent postoperative hemorrhage. It is doubtful that with the cystic artery and its larger branches this would always be sufficient. . . . The coagulum is likely to loosen sooner than the tie." The fact is that *I never coagulate the cystic artery* but always ligate it. Again, *the coagulum never loosens* in undrained, properly performed operations but becomes absorbed.

6. Careful scrutiny of the sixteen cases recorded by Whitaker forces the conclusion that unless one is equipped with a thorough knowledge of the principles underlying electrosurgical methods he had better hold on to the time-honored cold scalpel in surgery of the gallbladder.

MAX THOREK, M.D., *Professor of Surgery,*
Cook County Graduate School
of Medicine, Chicago.

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A CRITICISM OF AN EDITORIAL ON "POLIO VACCINES"

Editor, *New England Journal of Medicine,*

The *Journal* of October 3 carries an editorial headed "Polio Vaccines". In referring to vaccines, it says, "Thousands of mothers beg for its trial, and the supply of vaccine is limited. Before we again let our eagerness confuse our reason, it may be well to consider some of the facts in the matter."

The editorial then proceeds to make statements that are incorrect and not in accord with the latest work done on "polio" vaccine. The information thus conveyed is incorrect, misleading and not up-to-date.

The editorial states, "If we remember their (Harvard Infantile Paralysis Commission) laboratory experiences correctly, it would seem that the injection of subinfective amounts of living virulent virus, repeated many times, failed to produce sufficient active immunity to protect a susceptible monkey against subsequent inoculation with a fatal dose of virus."

Flexner and Lewis in 1910[1] reported the successful vaccination of monkeys with subcutaneous and intracutaneous injections of living virus or mixtures of virus and immune serum.

In 1927, Aycock and Kagan[2] did a great deal of work on monkeys, with a virus attenuated with phenol, glycerine and drying. They found that about 25 per cent of the animals were protected; but in another 25 per cent infection developed from the vaccine. In 1933, Kolmer and Rule[3] succeeded in vaccinating one monkey partially and two completely, out of a series of six monkeys by subcutaneous and intracutaneous injections of 2 per cent poliomyelitic cord in 10 per cent sodium ricinoleate. In 1934[4], Kolmer and Rule vaccinated 18 Macacus rhesus monkeys successfully against acute infantile paralysis with subcutaneous and intracutaneous injections of vaccine of living but attenuated virus. The vaccine was composed of 4 per cent suspensions of poliomyelitic monkey spinal cord in sterile 1 per cent solution of sodium ricinoleate.

These 18 monkeys were all immunized without the slightest ill effect and protected completely against infection following the intracerebral injection of about eighteen minimal infective doses of virus given under ether anesthesia about one month after the last dose of vaccine. The disease developed in the unvaccinated controls in from five to nine days after inoculation.

In 1934, Brodie[5] showed by work on monkeys:

1. A single subinfective intracutaneous inoculation of active poliomyelitis virus containing cord tissue gives rise to an immunity which appears at or soon after the sixth day and reaches its height by the twentieth day.
2. The immunity lasts a considerable time, being still present a year later.
3. Two doses of active cord tissue given at ten to twenty day intervals give rise to a higher degree of immunity than exists after mild or severe forms of the disease.

Both Kolmer and Brodie tried many methods of making a vaccine, safe for human beings; Brodie finally developing a vaccine from a formaldehyde treated monkey cord; Kolmer developing his from monkey cord using sodium ricinoleate as an attenuating agent.

The Brodie vaccine is a formaldehyde killed virus, the Kolmer, a living, attenuated virus.

After Kolmer had successfully vaccinated 18 monkeys and protected them against injections of virulent polio virus, without any ill effects whatsoever from the vaccine; he felt sure enough of his vaccine to start human work.

He proceeded to do this human work on himself and his co-worker, Anna Rule. First, he took some of his own serum, mixed it with living virus, and

after letting it stand for a time injected it into monkeys intracerebrally. He did the same with the serum of Anna Rule, and for controls used salt solution plus the same polio virus. All of the animals developed polio. He then proceeded to vaccinate himself and his associates. Two weeks after the third dose, he performed the serum neutralization tests again. Monkeys were injected as before with his serum plus polio virus, another group with her serum plus polio virus, and the control group with salt solution plus the polio virus. The Kolmer serum and the Rule serum absolutely protected their monkeys from any illness. The controls developed polio.

He had thus proved that the vaccine had developed in their blood some antiviral substance which protected the monkeys.

The work of both Kolmer and of Brodie has been carefully checked and controlled. I am referring more to the work of Kolmer because I am more familiar with it and have used his vaccine.

Kolmer[6] next proceeded to administer the vaccine to 25 children. Both before and after the vaccination, serum neutralization tests were done to test for antiviral substance in the blood.

These serum neutralization tests were done by mixing 0.5 cc. of serum with 0.5 of 10 per cent suspension of virus. This mixture was allowed to stand about two hours in a water bath at 37° C. and then injected intracerebrally into monkeys under ether anesthesia. For controls, 0.5 cc. of sterile saline was used with 0.5 cc. of virus, let stand in the water bath and used in the same manner.

There were no ill effects from the vaccine except slight fever in some of the children, 100.2° the highest, and a few slightly sore arms.

Of fifteen children showing no antibody before vaccination 75 per cent showed sufficient antibody following vaccination to protect the monkey. Serum of the ten children showing the presence of antiviral substance in their blood before vaccination showed much more after vaccination as their serum now protected the monkeys against an intracerebral injection of 50 per cent suspension of virus.

The serum neutralization test on monkeys[6] has so far proved the only test of value in determining whether the blood of the individual contains antibodies. Serum colloidal gold, complement fixation, precipitation[7] and skin tests[8] have been shown to be without value.

"The question of fundamental importance," says Kolmer, "is whether antibody in the blood of human beings for monkey passage virus is protective against human virus."

About 30 per cent of 126 cases of persons recovering from polio showed no antibody in their sera by monkey virus tests. This report is from the added results of eight different observers, and suggests that immunity to the disease can be present without demonstrable amounts of antibody in the blood.

Kolmer states: "These results indicate the possibility that antibody in the blood of human beings for monkey passage virus may not protect against human virus and if such should subsequently be proved it would render the present serum neutralization test worthless as an index or measure of resistance to poliomyelitis among human beings."

However, Kolmer has found that antibody developed in children by three doses of Kolmer vaccine has completely neutralized a human virus from the 1934 California epidemic.

The editorial proceeds to call attention to a question of simple arithmetic and gives an equation involving monkeys. Mathematics is an exact science; but this mathematical problem is based on a false premise.

To quote the editorial: "Since, for the production of vaccine by present methods, one monkey furnishes at the most, sufficient vaccine for ten injections, then each single injection would require one-tenth monkey."

Kolmer hoped to be able to prepare vaccine from brain tissue so that a greater amount would be available; but experiments showed that virus was absent or in insufficient amounts in brain tissue of monkeys dying of polio; so that the source of supply is limited to monkey spinal cord.

However, Kolmer states[6] that the spinal cord of one monkey furnishes about 150 cc. of vaccine which is sufficient for the immunization of from forty to seventy-five children depending on age and dosage. As three doses are required for each immunization, each single injection would require from one one-hundred-and-twentieth to one-two-hundred-and-twenty-fifth monkey instead of one-tenth.

Of course, it would be impossible to do monkey neutralization tests on all cases before and after vaccinations. It is practical, however, to do such tests on a percentage of the whole group vaccinated, and thus the results could be roughly figured out.

Kolmer's work indicates that the majority of children under ten years of age are devoid of antiviral antibody and probably susceptible.

Because of the fact that polio is a disease that hits only a small percentage of the population and because the disease rarely hits any locality in the same degree two successive years, it will take a long time to prove positively the value of any preventive measure.

So far[9] there is no knowledge of anyone carrying antibody in the blood, as shown by serum neutralization tests, developing infantile paralysis; but as yet only 318 sera have been reported on. On the other hand, polio has occurred in individuals known to have no antibody in the blood prior to the disease, and Kolmer has had one such case.

There is no doubt that all physicians are reluctant to inject a living though attenuated virus into human beings. In a letter dated September 3, Kolmer states that he and his associates have vaccinated over 6,000 cases—"only mild reactions developed, or none at all." Immunity develops very rapidly in at least 90 per cent.

My own experience is limited to twenty cases. No monkey neutralization tests were done and therefore the cases are of little value. However, it is of interest to note that not one of the cases showed the least constitutional reaction and the local reaction was so slight as to be unimportant.

The editorial ends, "But we hope that our eagerness will be tempered with reason and that any trial of any preventive or curative agent will be conducted with full and rigid controls, so that we may be spared the disappointment that has too often followed some of our earlier attempts."

I should like to point out that the work of both Kolmer and Brodie[10] has been carefully done and controlled so far as humanly possible.

It is best that we move forward rapidly in an endeavor to prove or disprove the value of the vaccine. It is true that we want to be spared the disappointment if it should fail; but it would be better to risk that disappointment than to proceed too slowly and cautiously, and later awake to find that we have been delaying in the use of a valuable preventive measure.

It would seem logical to select two adjacent primary schools, in several of our cities where epidemiological studies suggest that an infantile paralysis epidemic may occur next year. In the spring vaccinate the children in one school; use the other as a control. School has always adjourned for the summer before the disease becomes prevalent, and the children in adjoining districts would be in contact with each other during the summer. In this manner, the practical value of the vaccine could be determined in the shortest period.

JOHN F. CASEY, M.D.

475 Commonwealth Avenue,
Boston, Mass.,
October 9, 1935.

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EDITORIAL NOTE: The editorial on "polio" vaccines which appeared in the *Journal* of October 3, 1935, asks that the eagerness with which we grasp at any proffered solution of the poliomyelitis problem be tempered with reason. The writer of this editorial feels that merely recommending the trial of a vaccine (even though no assurance of efficacy be given) will be seized upon as a "cure" in view of the recurring surges of alarm. He would have wished for a more convincing demonstration of the efficacy and safety of the vaccine in the experimental animal before it was released for human use, and rightly goes on to point out some of the statistical difficulties in arriving at a prompt answer to the question of the efficacy of the vaccine.

Dr. Casey, on the other hand, in his criticism of the editorial, seems to accept the experimental evidence proffered by the proponents of the vaccine as sufficient justification for its human use, urging that we move forward rapidly in testing it out on children. Unfortunately, the scheme which he proposes to accomplish this—testing out the vaccine in communities shortly to be visited by infantile paralysis—would not be easy of fulfillment.

His own experience with twenty individuals, none of whom showed any general or local reaction to the vaccine, has little bearing on the main question involved, namely, whether the vaccine is a practicable means of preventing infantile paralysis.

IS LYMPHOCYTIC MENINGITIS SYMPTOMATIC OF A SYSTEMIC DISEASE?

October 25, 1935.

Editor, *New England Journal of Medicine*,

Your editorial entitled "Acute Lymphocytic Meningitis" which appeared in the *Journal*, September 19, 1935, is of considerable importance and the subject is of particular interest to me.

In 1929 when Viets and Watts[1] first reported the syndrome of "acute aseptic meningitis," I was fortunate enough to follow very closely a case with many similar characteristics at the Boston Psychopathic Hospital. This patient showing cerebral symptoms of an organic nature was found to have the typical signs and the blood picture of infectious mononucleosis commonly known as "acute glandular fever," originally described by Longcope[2] in 1922. Because of the uniqueness of the condition this case was reported in detail and published by Dameshek and myself[3] in the *Journal* in 1931 under the title of "Involvement of the Central Nervous System in a Case of Glandular Fever." Reference was made in that article to the earlier communication of Viets and Watts regarding acute lymphocytic meningitis and it was stated that "it is possible that the increase of lymphocytes in the spinal fluid (in such cases) may be associated with a lymphocytosis in the blood although as yet this has not been demonstrated." It was further stated that "the concept of an aseptic meningitis is at best a vague one and almost as all-inclusive as the term encephalitis." The concept was emphasized in that article that these conditions were symptomatic of some systemic disease and the suggestion was made that acute lymphocytic meningitis and infectious mononucleosis might be one and the same disease.

During the past few years a number of reports have appeared in the literature concerning the occurrence of this syndrome, variously called aseptic, idiopathic, epidemic and acute lymphocytic meningitis. So far as I am aware there have been no detailed blood studies in such cases and, therefore, it cannot be denied that there may be an etiological

relationship between acute lymphocytic meningitis and infectious mononucleosis. At the same time typical cases of the latter disease have been seen not infrequently and among these there have been no observations on the spinal fluid.

Considerable interest in the subject has developed recently as the result of some experimental investigations on the etiology of acute lymphocytic meningitis. The work of Armstrong[4] of the United States Public Health Service and of Rivers and Scott[5] of the Rockefeller Institute indicates that a filtrable virus is the causative agent of this disease, tending to show that the disease is a clinical entity.

It seems to me that the crux of the whole problem lies in the larger concept of various systemic diseases, of known and unknown causes, giving rise to changes in the central nervous system. I am inclined to believe that such diseases as infectious mononucleosis, still of unknown etiology, may produce cerebral changes, and conversely so-called acute lymphocytic meningitis like certain of the encephalitides, is merely symptomatic of some as yet unknown systemic disease.

Very truly yours,

SAMUEL H. EPSTEIN, M.D.

475 Commonwealth Avenue,
Boston, Mass.

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ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

Editor, *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of September 5 the following have been accepted:

Abbott Laboratories
- Procaine Hydrochloride — Abbott Tablets, 1.14 grains (0.07 Gm.)
- Procaine Hydrochloride —Abbott Tablets, 2.28 grains (0.15 Gm.)
- Ampoules Bismuth Subsalicylate with Butyn—D. R. L., 1 cc.

Robert A. Bernhard
- Saf-T-Top 5% Ferric Chloride in 50% Glycerine, 2 cc. and 15 cc.
- Saf-T-Top Isopropyl Alcohol, 98%, 2 cc. and 15 cc.
- Saf-T-Top Mercurochrome 2 per cent Solution, 15 cc.
- Saf-T-Top Mercurochrome 2 per cent in 25 per cent Glycerine, 2 cc. and 15 cc.
- Saf-T-Top Tincture Metaphen, 2 cc. and 15 cc.

Lee Laboratories
- Rabies Vaccine, Semple Method (Lee)

Lederle Laboratories, Inc.
- Scarlet Fever Streptococcus Antitoxin, "Globulin-Lederle-Modified"
- Refined Diphtheria Toxoid (Alum Precipitated) —Lederle, 0.5 cc. vial packages

Wm. S. Merrell Co.
- Viosterol in Oil—Merrell, Sperti Process
- Ampules Diothane 0.5% in Solution of Sodium Chloride 0.6%, 6 cc.

Winthrop Chemical Co., Inc.
- Salvarsan, 1.2 Gm. tubes

The following products have been accepted by the Council for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N. N. R. (New and Nonofficial Remedies, 1935, p. 445):

Robert A. Bernhard
- Saf-T-Top Tincture Iodine, U.S.P., 2 cc. and 15 cc.
- Saf-T-Top Tincture Iodine, U. S. P., 3½%, 2 cc. and 15 cc.

Yours sincerely,

PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

RECENT DEATHS

TUCKER—WILLIS LEROY TUCKER, M.D., of Hinsdale, Massachusetts, died at his home, November 7, 1935. He was born in 1872, graduated from the University of Illinois College of Medicine in 1893, and had practiced in Hinsdale for many years.

He served in the World War and had been a member of the board of selectmen and of the school committee in Hinsdale.

ANDREWS—FREEMAN W. ANDREWS, M.D., of 50 Peterboro Street, Boston, died at the Forest Hills Hospital, November 8, 1935.

He was born in Boston, the son of Clifford F. and Addie (Appleton) Andrews, in 1896.

He first qualified as an osteopathic practitioner and is recorded as a physician in the Directory of the American Medical Association but without a record of having an M.D. degree.

He served as official physician to the Back Bay Police Station. He is survived by his widow, Mrs. Audrey H. Andrews, and a daughter.

OBITUARY

RESOLUTIONS IN COMMEMORATION OF DR. WALTER A. POLLANO

ADOPTED AT THE NINETY-FOURTH QUARTERLY MEETING OF THE ESSEX NORTH DISTRICT MEDICAL SOCIETY

Whereas, Dr. Walter A. Pollano died suddenly on June 25, 1935, at his home at 238 Haverhill Street, Lawrence, Mass., from a heart attack.

Dr. Pollano was born in Sessa Aurunca, Italy, on April 6, 1879, and he came to Lawrence when he was twenty-one years old. He was educated in Italy

where he attended the University of Naples. In 1915 he became a registered pharmacist and from that time was actively interested in one of the Lawrence drug stores. He later studied medicine and received his degree of M.D. from the Middlesex College of Medicine and Surgery in 1922. He served his interneship at the Lying-In Hospital in New York. Dr. Pollano was affiliated with the American Medical Association, and the Massachusetts Medical Society becoming a member in 1928. He was also an active member of the Greater Lawrence Medical Association.

Whereas, in the death of Dr. Walter A. Pollano, the Massachusetts Medical Society has lost an honored and efficient member, and one held in high esteem and,

Whereas, we keenly feel our loss, and we desire to express some appreciation of his high qualities, his loyalty to his profession, and his friends and,

Whereas, we desire to express our heartfelt sympathy to his widow and his two small children,

Therefore, be it resolved that this resolution be entered upon the records of the Society, and that copies be sent to his widow, and to The *New England Journal of Medicine.*

Signed:

N. F. DeCesare, M.D.
N. J. Scarito, M.D.
Joseph A. Sinclitico, M.D.

NOTICES

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, November 15, 12-1, Cheever amphitheatre.

Dr. William R. Morrison, Associate Professor of Surgery, Boston University School of Medicine, will present the following cases:

1. Intestinal obstruction, with ileostomy.
2. Fracture of both bones of forearm.
3. Hypertrophied prostate with multiple urinary calculi.

Physicians and medical students are invited.

THE EDWARD K. DUNHAM LECTURESHIP

In 1923 there was founded in memory of Doctor Edward K. Dunham (M.D., Harvard, 1886), the Edward K. Dunham Lectureship for the Promotion of the Medical Sciences. Among the useful purposes for which the Foundation was established was that of binding closer "the bonds of fellowship and understanding between students and investigators in this and foreign countries." The lecturers are chosen from "eminent investigators and teachers in one of the branches of the Medical Sciences, or of the basic Sciences which contribute toward the advance of Medical Science in the broadest sense." The lectures, which are given annually, are "free and open to the faculty and students of the Harvard Medical School and College, and all other interested professional persons who may profit by them."

The Faculty of Medicine of Harvard University has issued invitations to attend three lectures to be given under the Edward K. Dunham Lectureship for the Promotion of the Medical Sciences on "Recent Studies on the Functions of the Hypophysis."

Friday, November 22, "What We Have Learned from the Toad Concerning Hypophyseal Functions."

Monday, November 25, "Metabolic Functions of the Hypophysis."

Wednesday, November 27, "The Hypophysis and Diabetes."

At five o'clock at the Harvard Medical School Amphitheater, Building C, by Bernard A. Houssay, M.D., Professor of Physiology and Director of the Institute of Physiology, University of Buenos Aires.

MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, November 21, in the amphitheater of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian.

UNITED STATES CIVIL SERVICE EXAMINATIONS

The United States Civil Service Commission has announced open competitive examination as follows:

Public Health Consultants

Applications for various grades of public health consultants, and for public health research assistant, must be on file with the U. S. Civil Service Commission, Washington, D.C., not later than November 25, 1935.

Optional subjects for the consultant positions are maternal and child health, general public health practice, and orthopedics. The specialty for the research assistant is maternal and child health.

Vacancies in these positions in the Children's Bureau, Department of Labor, and in the U. S. Public Health Service, Treasury Department, both in Washington, D. C., and in the field, will be filled from these examinations.

Social Economists

Applications for various grades of social economist positions, Children's Bureau, Department of Labor, must be on file with the U. S. Civil Service Commission, Washington, D.C., not later than November 18, 1935.

Optional subjects are: Child welfare administration; social service for physically handicapped children; research and demonstration in the prevention and treatment of juvenile delinquency; and research in problems of child dependency and neglect, or social provision for crippled children.

Certain education and experience are required.

Full information may be obtained from the Secretary of the United States Civil Service Board of Examiners at the post office or customhouse in any city which has a post office of the first or the second class, or from the United States Civil Service Commission, Washington, D.C.

REPORTS AND NOTICES OF MEETINGS

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The Worcester North District Medical Society held the most largely attended and most enthusiastic meeting in its history at the State Colony in Gardner Wednesday, October 23. The Society was signally honored by the presence of Dr. Charles E. Mongan, President of the Massachusetts Medical Society; Dr. Alexander S. Begg, Secretary of the Massachusetts Medical Society; Dr. William Morrison, Chairman of the Committee of Arrangements for the next annual meeting of the Society; and Dr. M. A. Tighe, Chairman of the Committee on Social Legislation and Insurance, a subcommittee of the Public Relations Committee of the Massachusetts Medical Society. There were about one hundred and forty present. Dr. George P. Norton, President of the Worcester North Society, introduced the speakers and referred to the great honor conferred on the Society by the distinguished guests.

Dr. Mongan showed an intimate knowledge of the history of the local society and recalled that the late Dr. Alfred O. Hitchcock of this city had been president of the state society in 1869 and that Dr. F. H. Thompson, Sr., of this city had been a vice-president. He paid a fine tribute to the efforts of the late Dr. Alfred H. Quessy in defeating the Sheppard-Towner bill.

Dr. Mongan said that the state society needs the help of all medical men. The doctor must become politically minded, he said. He no longer can refuse to do his share in great movements, many of which cannot be successfully worked out without the coöperation of the physicians.

He argued that conditions in Massachusetts are different from those in many other states and we should work out our own problems.

"The idealist wants to abolish poverty, but it cannot be accomplished," he said. "Those who would attempt to carry out their plans along these lines forget that the spirit of America is based on manhood and the acceptance of opportunities. The entire movement came from Europe and from Oriental countries where all reliance is placed in the head of the country, thereby stifling initiative.

"But even in this country the morale has broken down to some extent and persons who a few years ago would spurn government aid now consider it as one of their rights. England has a fine sense of propriety and the English are willing to give for their country. The medical men can do much to check the present tendency of affairs for they go from home to home, are asked advice and frequently give it."

He asked the members to go to their state representatives or congressmen to prevent bad legislation.

"There is no need of compulsory health insurance in this state, for the doctors have always given the highest type of service. There is a difference between public health and private health. Compulsory health insurance means that everyone with a salary of $3000 per year or less must have health insurance, the employer and employee each to pay 40 per cent and the state 20 per cent of the cost."

He called for concerted action by the public to defeat the enforcement of the provisions of the compulsory health insurance act.

Dr. Tighe said that the two influences aiding the passage of the compulsory health act were the report of a committee of four appointed by the administration, no one of which committee was a doctor, and the depression.

"This latter was duck-soup for the idealists," he said. "It was what they were waiting for. As a result, President Roosevelt appointed a national security committee whose task was to cure all the social evils. Since no doctors were on this committee the secretary of labor, Miss Perkins, appointed her own group, three of whom were doctors but they are sick of their jobs already.

"The delegates of the American Medical Association oppose this feature. The Councilors of the Massachusetts Medical Society have joined them in their opposition and the Public Relations Committee has set out on a program of publicity and education of the people to mould public opinion against this iniquitous bill."

Dr. Alexander S. Begg, Secretary of the State Medical Society, gave a brief talk, congratulating the members on the splendid attendance, despite the heavy rainfall.

Dr. Morrison, Chairman of the Committee of Arrangements of the next Annual Meeting of the State Society to be held in Springfield, June 8, 9, and 10, gave a résumé of the plans.

Dr. Thomas R. Donovan, Supervisor of the Board of Censors, announced that the next meeting of the Censors would be held at the Burbank Hospital, Thursday, November 7, at 4 P.M.

Dr. Edward A. Adams told of the courses given in postgraduate instruction in the district. A motion by Dr. Walter F. Sawyer was passed whereby the cancer clinics of the district will be operated separately.

A splendid buffet lunch was served and a vote of thanks given to the speakers.

FRANCIS W. MCMURRAY, M.D., *Secretary.*

MASSACHUSETTS PSYCHIATRIC SOCIETY

The eleventh annual meeting of the Massachusetts Psychiatric Society was held on October 29, 1935, at the Parker House, Boston.

The following officers were elected for the coming year:

President, Dr. Charles E. Thompson.

Vice President, Dr. Oscar J. Raeder.

Secretary-Treasurer, Dr. W. Franklin Wood.

Councilors, Dr. C. Macfie Campbell, Dr. Joseph E. Barrett.

A large attendance listened with keen interest to Dr. George Sarton's illuminating address on "Genius." He showed in the development of his subject the necessity of discounting the psychopathic tendencies in the various types of genius. He emphasized the point that the now popular type of "debunking" biography often lays too much stress on these psychopathic traits and does not sufficiently feature the extraordinary talent of the particular genius, for which alone he is interesting. He said that all the traits of such a great man can be imitated except his genius—that is peculiarly his own.

OSCAR J. RAEDER, M.D., *Secretary-Treasurer.*

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

A stated meeting of Essex South District Medical Society was held at the Beverly Hospital, November 6, 1935.

An interesting and instructive clinic was held at 5 P.M., followed by a dinner at 7 P.M. The guest speakers, at the evening meeting, were Dr. Charles E. Mongan, President of the Massachusetts Medical Society, Dr. Michael A. Tighe, Chairman of the Committee on Social Legislation and Insurance, a subcommittee of the Public Relations Committee of the Massachusettts Medical Society and Dr. Alexander S. Begg, Secretary of the Massachusetts Medical Society.

Dr. Mongan reminded the members of their splendid professional heritage and of their consequent responsibility for good influence in the community and interest in the community welfare. His appeal was for careful appraisal of so-called "economic security"; supporting the good in it and preventing the bad.

He also appealed for the profession, as a whole, to become professionally and politically conscious for the benefit of the country.

Dr. Tighe, of Lowell, Mass, reviewed briefly the history of "Compulsory Sickness Insurance" in England and Germany.

Citing the vote of the American Medical Association to disapprove all Compulsory Sickness Insurance and the approval of this stand by the Massachusetts Medical Society, he called especial attention to the fact that the Massachusetts Medical Society had initiated a campaign of public education concerning Compulsory Sickness Insurance.

He expressed his absolute faith in public opinion when rightly informed and was confident that a high degree of interest in public good existed among the laity and that the press could always be relied upon correctly to interpret this interest, without political bias.

Dr. Begg complimented the Staff of the Beverly Hospital for the valuable clinic presented to the medical society and graciously offered the facilities of his office in any way that might benefit any district society. NATHANIEL POPE BREED, M.D., *Reporter.*

HAMPDEN DISTRICT MEDICAL SOCIETY

From eighty to ninety members and guests attended a meeting of the Hampden District Medical Society held at the Academy of Medicine, Springfield, October 29, 1935. The President, Dr. Theodore S. Bacon, presided.

The paper for the afternoon was on "Recent Advances in Medicine", by Dr. Reginald Fitz, of Boston.

Dr. Fitz touched rather briefly on the question of State Medicine, its possibilities and its disadvantages. He praised the high degree of efficiency attained by organized medicine in the American Expeditionary Forces in the World War. He cited diseases formerly considered rather hopeless in which recent advances have radically changed our attitude, e.g., arthritis, hypertension, especially of the functional type, and certain formerly obscure endocrine disturbances; also the anemias and cardiovascular-renal disease. He mentioned dietetics as a field in which marked advances had been made through careful observation and experimental work. He is of the opinion that the state of adolescence will yield a rich return if carefully studied. It is here that the apparent results of endocrine therapy may be misleading. Methods recently introduced for the clinical study of the peripheral circulation and of the blood and blood-forming organs in the anemias were mentioned and the present status of classification of the anemias, which fall roughly into well-defined groups and a borderline one, more or less composite. Some recent surgical methods developed in the treatment of conditions formerly regarded as entirely medical were referred to and the work of the Mayo and Lahey Clinics and of a surgical group in Michigan. Dr. Fitz believes that at no time has the medical profession been more alive and forward-looking than at present.

After a standing vote of thanks to Dr. Fitz the meeting adjourned and a buffet supper was served.

ANDREW PETERS, M.D., *Reporter.*

CLOVER HILL HOSPITAL

Lawrence, Mass.

The next medical meeting of the Clover Hill Hospital will be held in the reception room of the hospital at 161 Berkeley Street, Lawrence, on Thursday afternoon, November 21, 1935, at 4:30 P.M.

Speaker: George R. Minot, M.D., Professor of Medicine at Harvard University.

Subject: "The Etiology, Diagnosis and Treatment of Nutritional Deficiency Anemias."

The lecture will be illustrated with lantern slides. Discussion will follow. A clinical exhibition arranged by Lawrence physicians may be seen following the lecture. All physicians of Lawrence and vicinity are cordially invited to attend.

EDWARD MACK SMITH, M.D., *Chairman.*

THE BOSTON HEALTH LEAGUE AND THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

A Luncheon Meeting of the Boston Health League and the Massachusetts Central Health Council will be held at The Parker House, Boston, on Thursday, November 21, 1935, at 12:30 P.M.

Dr. Carl Buck, Field Director of the American Public Health Association, will speak on "New Horizons in Public Health Work".

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The New England Physical Therapy Society will meet at 8 P.M. on Wednesday, November 20, at the Hotel Kenmore, 490 Commonwealth Avenue, Boston.

PROGRAM

Treatment of Prostate Pathology. George C. Prather, M.D., Boston; George E. Percy, M.D., Salem.

Opening comments by DeWitt G. Wilcox, M.D., Newton, and William D. McFee, M.D., Boston, followed by general discussion.

Special dinner arrangements have been made for a table in the Empire Room of the Kenmore at 6:30 P.M. Members and guests are urged to take advantage of this social hour.

The council will meet at 6 P.M.

All members of the medical profession are cordially invited to attend.

WILLIAM D. MCFEE, M.D., *Secretary.*

41 Bay State Road,
Boston, Mass.

NEW ENGLAND HEART ASSOCIATION

Boston City Hospital
Amphitheatre of the Mallory Institute of Pathology
(Entrance at 784 Massachusetts Avenue)
Monday, November 25, at 8:15 P.M.

PROGRAM

1. Demonstration of Pathological Specimens.
2. Amyloid Heart. Dr. G. Kenneth Mallory.
3. The Mechanism and Treatment of Postural Hypotension. Dr. L. B. Ellis and Dr. Florence Haynes.
4. The Significance of Precordial Leads in Cardiac Infarction — Clinical-Pathological Correlation. Dr. James M. Faulkner.
5. Recent Observations on the Functional Properties of the Vascular System and on the Hemodynamics in Arterial Hypertension. Dr. Soma Weiss.
6. The Nature of the Peripheral Resistance in Arterial Hypertension with Special Reference to the Vasomotor System. Dr. Myron Prinzmetal and Dr. Clifford Wilson.
7. The Electrocardiogram in Bacterial Endocarditis as Contrasted with Rheumatic Carditis. Dr. Maurice S. Segal.
8. The Significance of Differential Venous Pressure Measurements. Dr. Eugene B. Ferris, Jr. Presented by Dr. Robert W. Wilkins.
9. Malignant Hypertension of the Pulmonary Circuit (?). Dr. Frederic Parker, Jr., and Dr. Soma Weiss.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, November 26, at 8:15 P.M.

PROGRAM

Presentation of Cases.

The Mechanism and Effects of Abdominal Compression in the Treatment of Pulmonary Tuberculosis. By Dr. Burgess Gordon, Associate Professor of Medicine, Jefferson Medical College.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

CARNEY HOSPITAL

CLINICAL MEETING

A Clinical Meeting of the Carney Hospital will be held at 8:30 P.M. on Monday, November 18.

PROGRAM

Blood Transfusion. By Dr. Lewis S. Pilcher. (Lantern Slides.)

The Routine Management of Diabetic Patients. By Dr. C. W. Finnerty.

Physicians and medical students are invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, NOVEMBER 18, 1935

Monday, November 18—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Blood Clinic. Dr. William Dameshek.

*8 P.M. Medical Historical Pageant. Boston Medical Library. 8 Fenway.

*8:30 P.M. Carney Hospital, Clinical Meeting.

Tuesday, November 19—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Back Strain—Sciatica. Dr. F. R. Ober.

*12 Noon. South End Medical Club, office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

7:45 P.M. Gardner Auditorium, State House, Boston. "The Child and the Modern World." Lawson G. Lowrey, M.D.

Wednesday, November 20—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Ward Cases. Dr. S. J. Thannhauser.

†12 M. Clinico-Pathological Conference. Children's Hospital.

1 P.M. Massachusetts Society for Mental Hygiene, Twentieth Century Club, Boston.

*8 P.M. New England Physical Therapy Society. Hotel Kenmore, Boston.

Thursday, November 21—

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. "Interviewing a Patient." Professor Elton Mayo.

12:30 P.M. The Boston Health League and the Massachusetts Central Health Council. The Parker House, Boston.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

5 P.M. Lecture on "The Care of the Patient," Harvard Medical School, Amphitheatre C.

Friday, November 22—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Ward Cases. Dr. S. J. Thannhauser.

12 M. Clinical Meeting of the Children's Medical Staff. Ether Dome, Massachusetts General Hospital.

5 P.M. The Edward K. Dunham Lectures. Harvard Medical School Amphitheatre, Building C.

Saturday, November 23—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Liver Disease. (Continued.) Dr. S. J. Thannhauser.

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

November 14—Harvard Medical School. Lecture on "The Care of the Patient," Dr. Arthur R. Crandell. Amphitheatre C at 5 P.M.

November 15—New England Roentgen Ray Society will meet at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, at 8:15 P.M.

November 15—Boston Floating Hospital Staff Meeting will be held at 12 noon.

November 15—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 1000.

November 18—Medical Historical Pageant. See page 993.

November 18—Carney Hospital, Clinical Meeting. See page 1003.

November 19—Lawrence Cancer Clinic, Lawrence General Hospital, One Garden Street, 10 A.M.

November 19—The South End Medical Club will meet at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 noon.

November 20—Massachusetts Society for Mental Hygiene will meet at the Twentieth Century Club, Boston, at 1 o'clock.

November 20—New England Physical Therapy Society. See page 1003.

November 21—Harvard Medical School. Lecture on "The Care of the Patient." Dr. David D. Scannell. Amphitheatre C at 5 P.M.

November 21—Medical Clinic at the Peter Bent Brigham Hospital. See page 1000.

November 21—The Boston Health League and the Massachusetts Central Health Council. See page 1003.

November 21—Clover Hill Hospital. See page 1002.

November 22, 25, and 27—The Edward K. Dunham Lectures. Harvard Medical School. See page 1000.

November 25—New England Heart Association. See page 1003.

November 26—Harvard Medical Society. See page 1003.

December 5-7—National Society for the Prevention of Blindness. See page 940, issue of November 7.

December 13—William Harvey Society. Beth Israel Hospital, 8 P.M.

DISTRICT MEDICAL SOCIETIES

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings are held on the second Tuesday of January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

November 26—Norwood Hospital at 8 P.M. Papers by the staff.

January 28, 1936—Hotel Kenmore at 8 P.M. Dr. Benedict F. Boland—"Cauterization of the Cervix Uteri Using Various Electrical Methods." Illustrated with lantern slides.

February 25, 1936—Massachusetts Memorial Hospitals at 8 P.M. Papers by the staff.

March 31, 1936—Hotel Kenmore, at 8 P.M. (Subject to be announced.)

May, 1936—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D.
1236 Beacon Street, Brookline, Massachusetts.

PLYMOUTH DISTRICT MEDICAL SOCIETY

November 21—State Farm.

January 16—Goddard Hospital. Subject and speakers to be announced later.

March 19—Plymouth County Sanatorium, South Hanson.

April 16—Brockton Hospital.

May 21—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.

SUFFOLK DISTRICT MEDICAL SOCIETY

December 11—Joint Meeting with the New England Heart Association at the Boston Medical Library. "Constrictive Disease of the Pericardium," Dr. Charles Sidney Burwell. Discussion: Dr. Edward D. Churchill and Dr. Paul D. White.

January 29, 1936—Joint Meeting with the Boston Medical Library at 8 Fenway. "Observations Around the World," Dr. Walter B. Cannon.

March 18, 1936—Meeting at the Boston Medical Library. "The Laboratory and Clinical Story of Fatigue," Dr. Arlie V. Bock and Dr. David B. Dill. Discussion: Dr. Donald J. MacPherson and Dr. Augustus Thorndike, Jr.

April 29, 1936—Annual Meeting at the Boston Medical Library. "The Treatment of Septicaemia," Dr. Champ Lyons. "The Pleurality of Scarlatinal Streptococcus Toxin," Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary,
FRANCIS T. HUNTER, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

November 20—Special meeting. See page 949, issue of November 7.

December 11—Wednesday evening. St. Vincent Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

January 8, 1936—Wednesday evening. Worcester City Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

February 12, 1936—Wednesday evening. Worcester State Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

March 11, 1936—Wednesday evening. Memorial Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

April 8, 1936—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

May 13, 1936—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.

BOOK REVIEW

New and Supplementary Facts and Figures About Tuberculosis. Jessamine S. Whitney. 46 pp. New York: National Tuberculosis Association. $.50.

This pamphlet, published in June, 1935, by the National Tuberculosis Association, is complementary to an earlier compilation of facts and figures about tuberculosis issued in 1931. It consists of forty-nine elaborate tables including a group of tables of great importance on tuberculous infection.

To those who are investigating and writing on the mortality and morbidity of tuberculosis and who need statistical information of any kind whatsoever on this subject, this booklet will prove a veritable mine of useful, accurate and up-to-date information.